Neonatal outcome in the CTG group

Neonatal outcome in the CTG group

(ST data blinded to attending staff)

Perinatal death

Case MAB 215

Date of delivery; [illegible]

Clinical data

Para 0, 41 weeks of gestation.
Spontaneous onset of labor.
Artificial rupture of membranes at 21:59h showing meconium-stained liquor.
Augmented labour
Mid cavity ventouse for non-reassuring fetal heart rate at 04:26.

Neonatal data

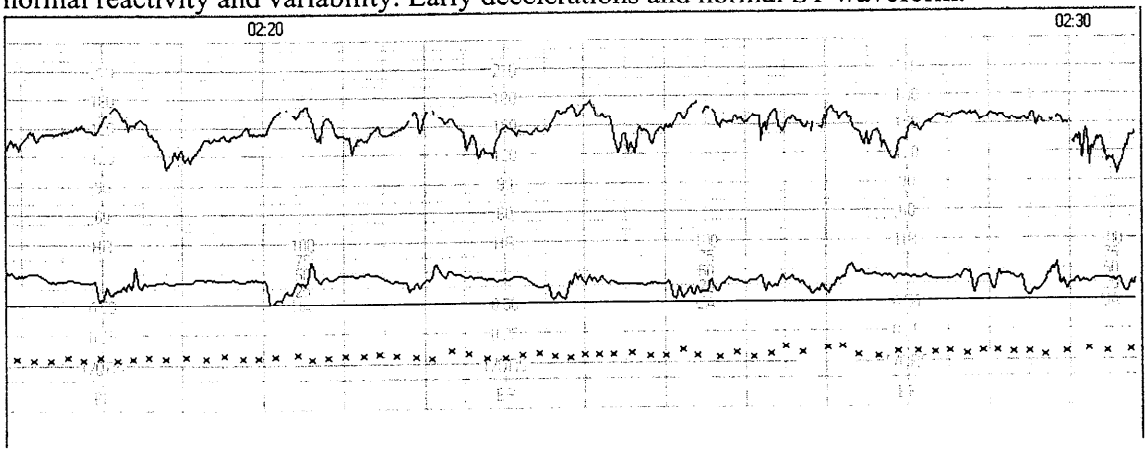
Male 4105 g
Apgar 0-2-3.
Cord artery: pH 6.77
PCO_2 14.29 kPa
BDecf 17.3 mmol/l

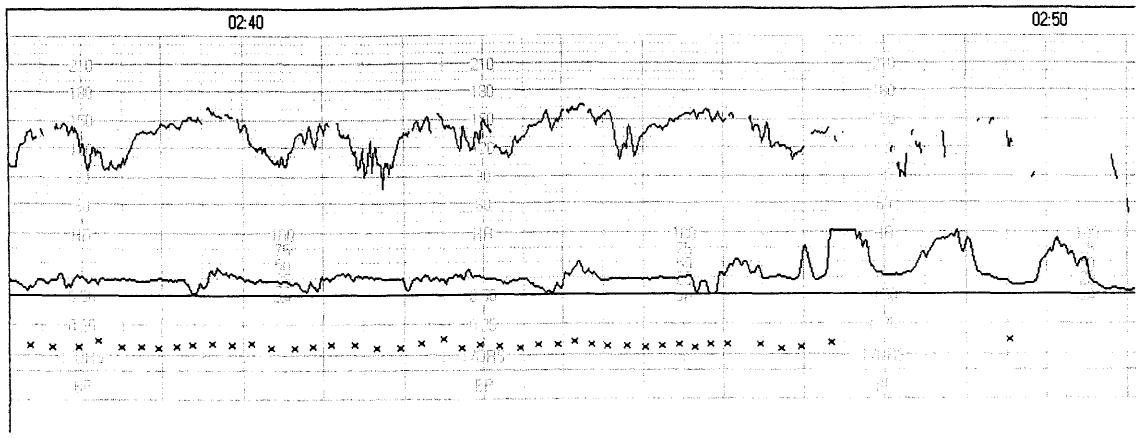
Neonatal outcome

Death occurred at 24h of age due to severe perinatal asphyxia with clinical signs of severe neonatal encephalopathy (coma, cardio-respiratory failure).

Assessment of the recording

Trace obtained during 1st stage of labor shows slight increase of baseline fetal heart rate with normal reactivity and variability. Early decelerations and normal ST waveform.





Comments

The STAN recorder was disconnected at 02:57 h (2 h and 29 min before delivery) for no obvious reason. The case was managed based on CTG-only information obtained from a standard CTG monitor thereafter.

Cases with severe neonatal encephalopathy (neonatal seizures and neuroradiological findings)

Case OEK 309

Date of delivery: [illegible]

Clinical data

Para 0, normal pregnancy, 40 weeks of gestation
Clear liquor, augmented labour, epidural analgesia.
Spontaneous onset of labour: [illegible] at 22:00h
Active pushing [illegible] at 18:45
NSVD at 19:10

Neonatal data

Female 3535 g
Apgar 4-6-6
Flaccid baby requiring assisted ventilation.
No cord acid-base data obtained.
Venous blood data at 34 min of age: pH 7.22
PCO_2 3.41
BDecf 15.3 mmol/l, 15 ml buffer given.
normal glucose (3.8 mmol/l),

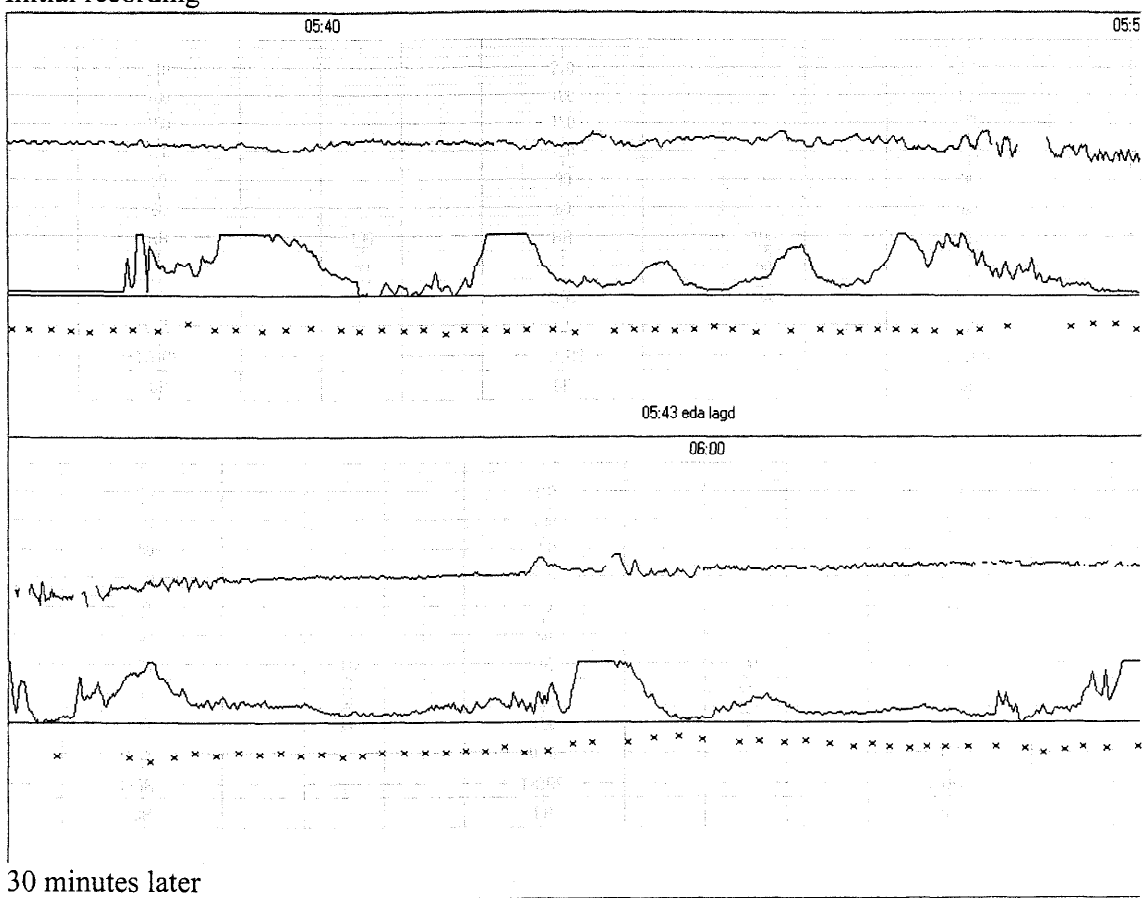
Neonatal outcome

Seizures during the first day and phenobarbiturate treatment. Computed tomography (CT) of the brain showed ischemic lesions in the occipital region with corresponding electroencephalographic findings. Three weeks in Special Care Baby Unit (SCBU).

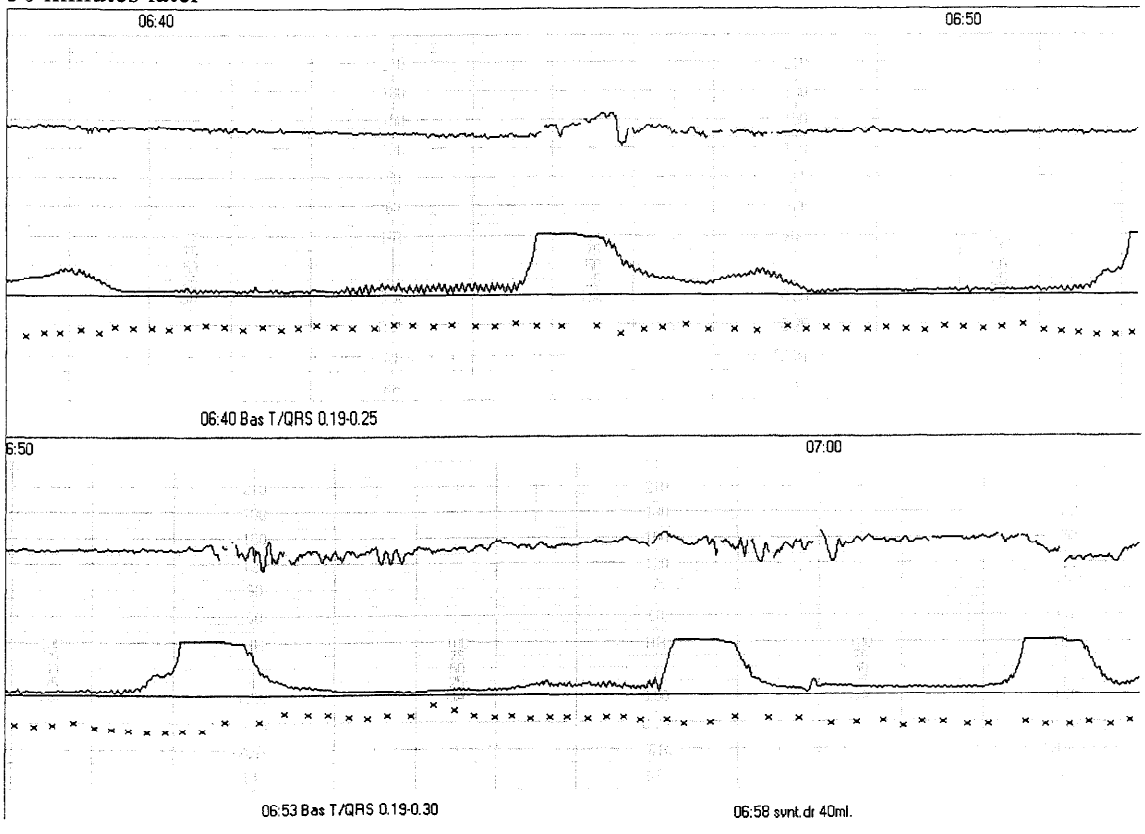
Assessment of the recording

FECG data were recorded between 05:17 – 19:06. Already at 05:50h, with the occurrence of the first FHR deceleration and immediately after the epidural (05:43), an episodic T/QRS rise was recorded (see Fig). At 06:40, after the onset of oxytocin infusion, a baseline rise in T/QRS occurred with further FHR decelerations; followed by an episodic T/QRS rise of 0.18. The baseline T/QRS rise continued for the next 5 hrs in association with late decelerations. Towards the end of the recording, the T/QRS ratio decreased with continuing abnormal CTG (late decelerations, increase in baseline FHR and reduced variability). Throughout the recording, the uterine contraction pattern showed signs of over stimulation as a result of augmentation with slow progress. According to the CTG+ST guidelines, intervention was indicated 740 minutes before delivery.

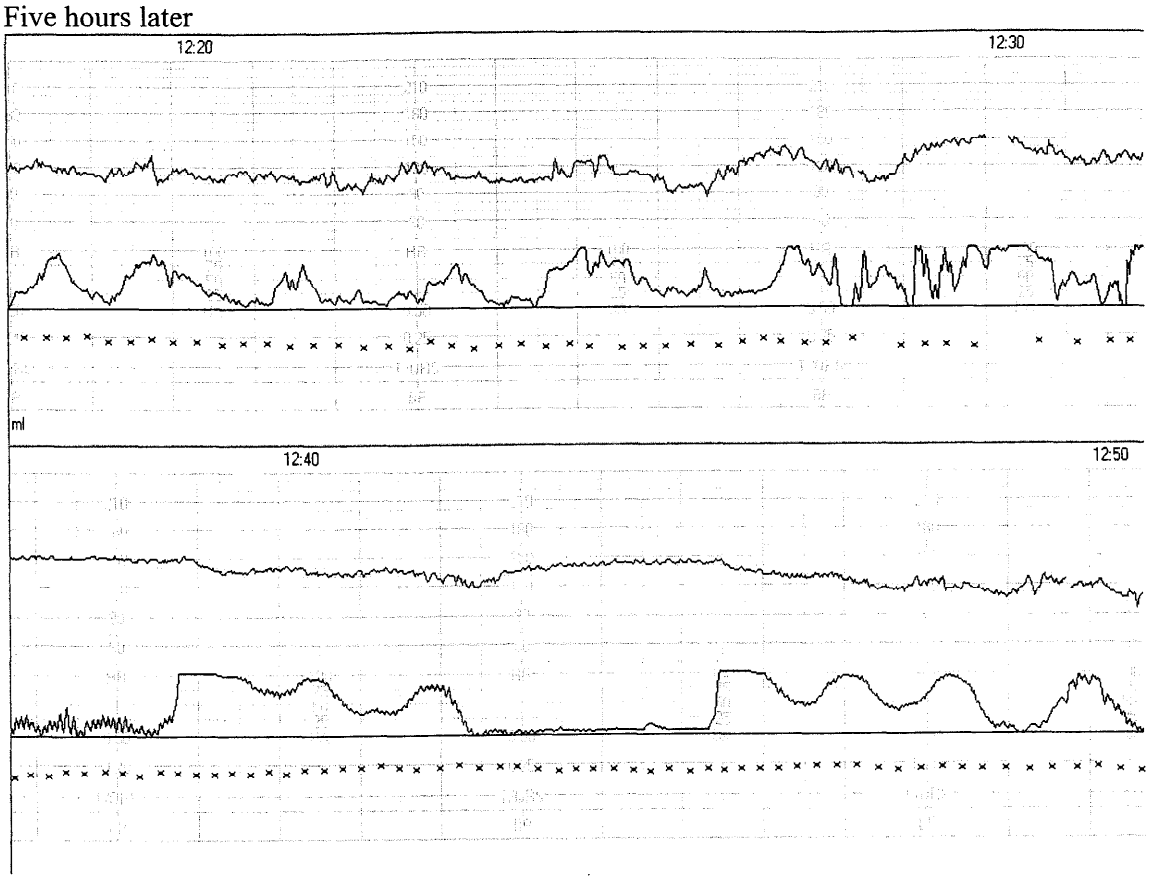
Initial recording



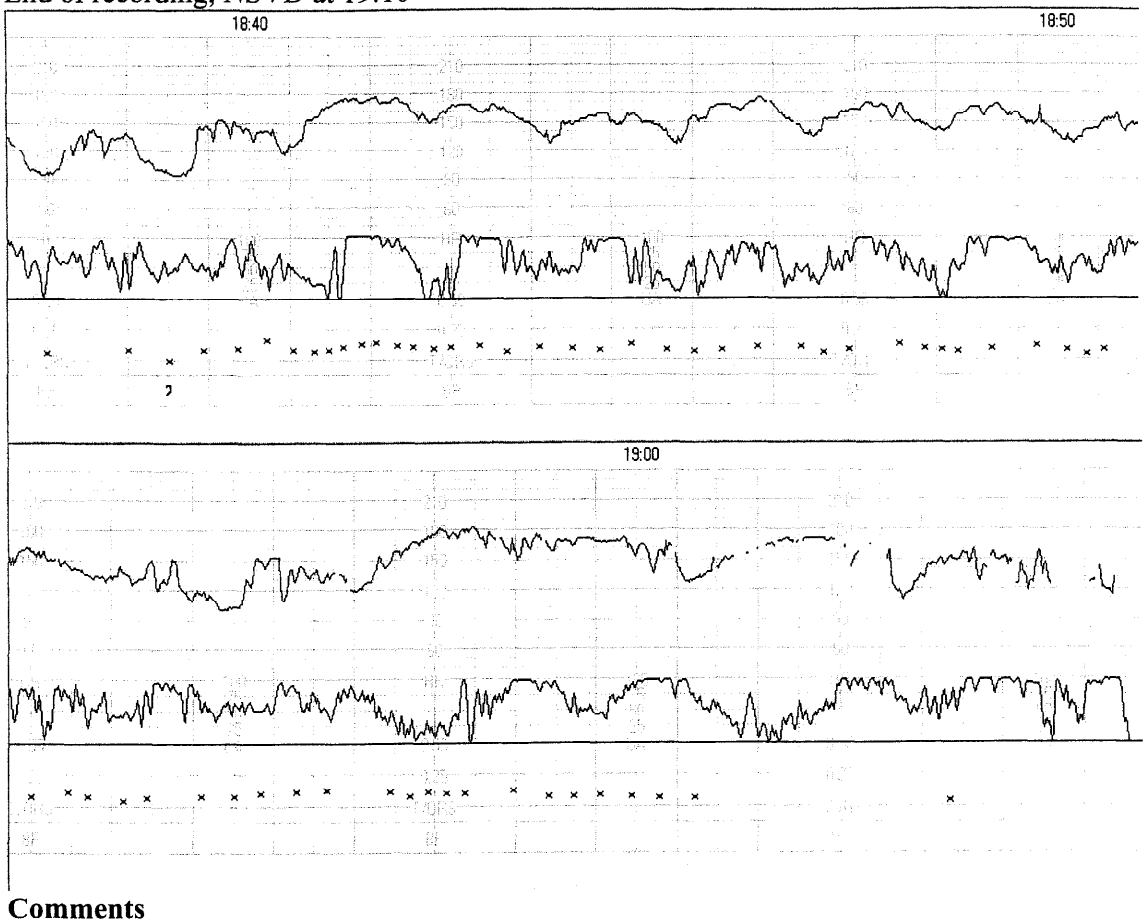
30 minutes later



Five hours later



End of recording, NSVD at 19:10



Comments

Significant ST events occurred during the first stage of labour, which by then already had lasted 8 – 9 hrs. A fetal scalp electrode was applied in conjunction with an epidural and augmentation of labor. It appears as if the first ST event was caused by the combination of the epidural and marked contractions, indicating a decreased placental and fetal reserve. The T/QRS ratio remained elevated until 2nd stage with continuing CTG abnormalities. Thus, there are reasons to believe this fetus had a diminished placental reserve with a long-lasting intrapartum hypoxia related to slow progress and uterine hyper-stimulation. The decrease of T/QRS during 2nd stage may be regarded as a sign of a decrease (down regulation) of fetal reactiveness due to chronic hypoxia.

Case OEH 379

Date of delivery: [illegible]

Clinical data

Para 0, normal pregnancy, spontaneous onset after 39 w
Clear liquor, epidural and oxytocin infusion.
Onset of labour at 14:00h
Active pushing at 22:06h
An operative vaginal delivery, mid cavity ventouse, was performed for fetal distress as indicated by a scalp-pH of 7.16 at 23:41.
Fetal scalp samples performed for non-reassuring fetal heart rate: increased baseline FHR, reduced variability, and variable decelerations.

21:52 pH 7.22 (preacidotic)
22:34 pH 7.22 (preacidotic)
23:02 pH 7.16 (acidotic)

The vaginal operative procedure was undertaken in theatre as a complicated vaginal operative delivery was anticipated. Complicated mid cavity ventouse also occurred where the ventouse was reapplied twice. After four extractions and with the support of externally applied pressure, the fetal head was delivered. Thereafter, shoulder dystocia occurred and delivery of the fetal body was delayed by 8-10 minutes.

Neonatal data

Male 4140 g
Apgar 1-2-3
Cord acid base was obtained from only one vessel: pH 7.14
PCO_2 9.62 kPa
BDecf 3.7 mmol/l.

Venous blood sample at 13 min age: pH 6.79
PCO_2 14.2 kPa
BDecf 16.5 mmol/l
lactate 17.1 mmol/l
blood glucose 11.7 mmol/l..

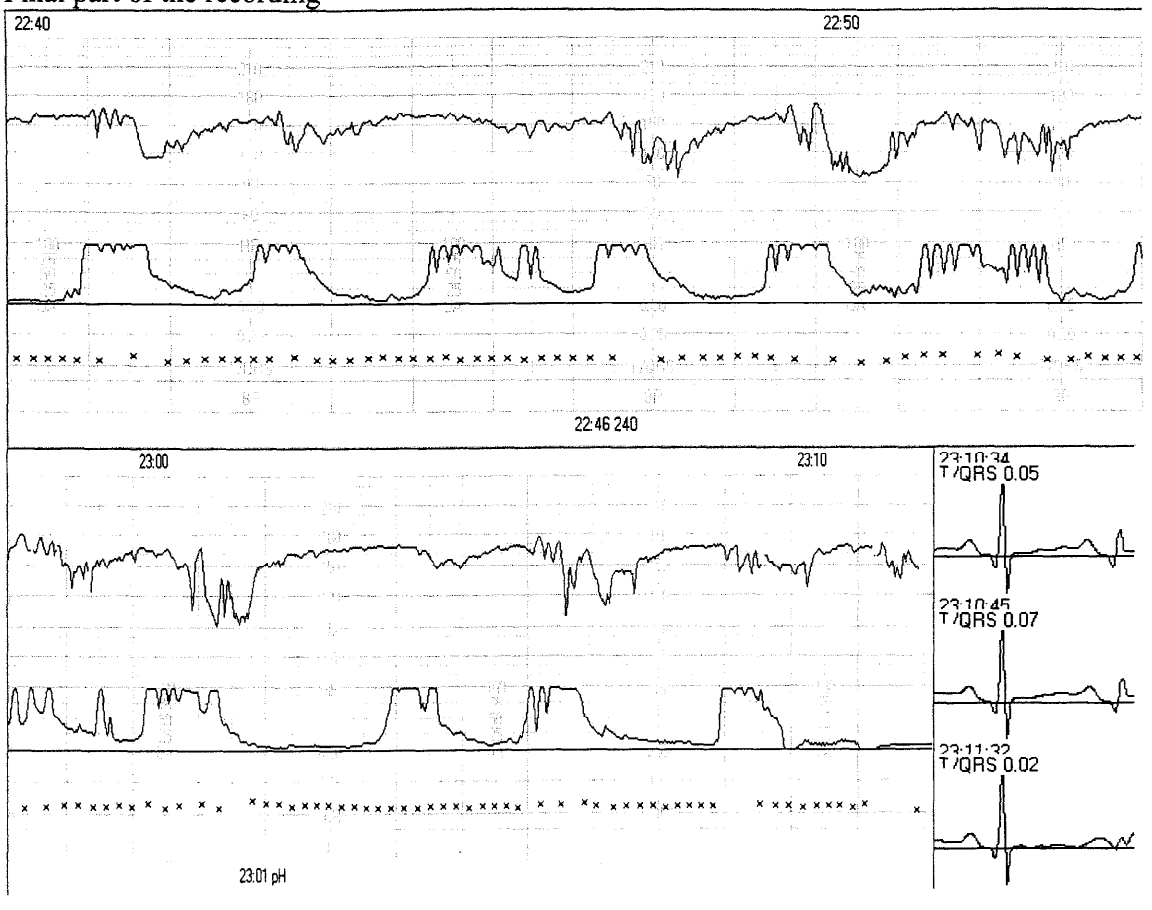
Neonatal outcome

The baby required immediate resuscitation with intubation, assisted ventilation and buffering. The baby suffered a subarachnoidal bleeding, damage to the brachial plexus and neonatal seizures. There were also signs of multi-organ failure with first day oliguria and increased liver enzymes.

Assessment of the recording

CTG showed tachycardia but a reactive pattern. Ventouse intervention was indicated by a scalp pH of 7.16 obtained during 2nd stage with active pushing.
There were normal ST waveforms throughout the recording.

Final part of the recording



Comments

The intervention was indicated because of a scalp pH of 7.16 obtained during active pushing, a situation where a decrease in pH due to a respiratory acidemia, would be more a reflection of the normal events of 2nd stage with short lasting cord compressions causing FHR decelerations and the accumulation of carbon dioxide. According to CTG+ST guidelines, intervention was not indicated at the time of the FBS.

The discrepancy between the cord acid-base and the marked metabolic acidosis detected postpartum could probably be explained on the basis of a "hidden" acidosis developing in the tissues (tissue trapping) and not reaching the vascular compartment until normal blood circulation was restored. A peripheral metabolic acidosis is not to be detected by analysis of stagnant blood in a compressed umbilical cord, but well after a restored circulation and washout of peripheral metabolites. The scalp electrode was disconnected 29 minutes before delivery.

Case LDA 23

Date of delivery: 

Clinical data
Para 0, 41 gestational weeks, normal pregnancy.
Spontaneous onset at 04:00h, augmented labour, epidural.
Active pushing:11:30h
Operative vaginal delivery, mid cavity ventouse at 11:54, for non-reassuring fetal heart rate.
Fetal scalp samples performed accordingly:

08:44	7.34 (normal)
09:38	7.28 (normal)
10:12	7.27 (normal)
10:50	7.20 (preacidotic)
11:17	7.30 (normal)

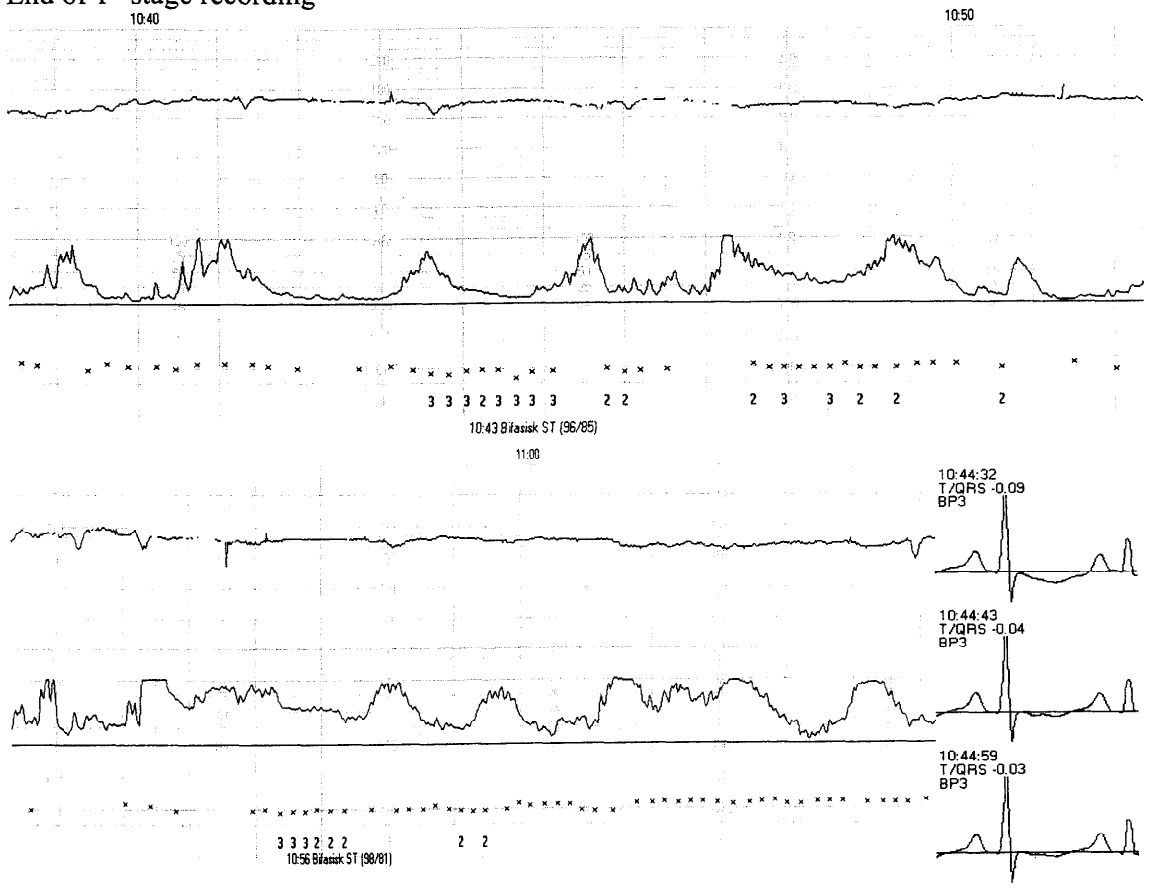
Neonatal data
Male 4020 g
Apgar 9-10-10

Cord artery:	pH 7.10
	PCO_2 6.07 kPa
	BDecf 13.7 mmol/l
Cord vein	pH 7.25
	PCO_2 4.92 kPa
	BDecf 4.4 mmol/l

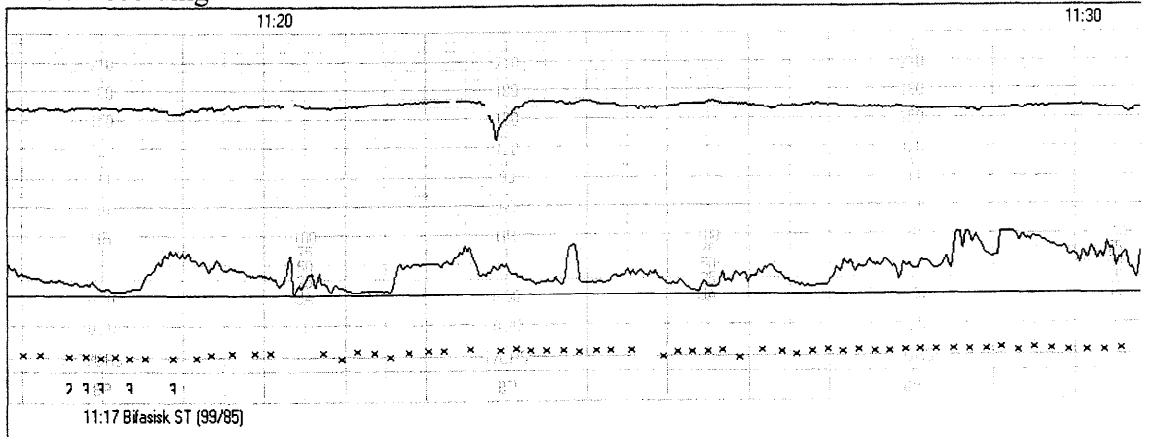
Neonatal outcome
Neonatal seizures occurred during the second day. Computed tomography of the brain on Day 2 showed subdural (tentorial) bleeding + cerebral ischemia in the left hemisphere. Magnetic Resonance angiography showed a most unusual finding of a dissection of the left carotid artery with signs of peripheral arterial embolisation causing arterial occlusion and cerebral ischemia. Abnormal electroencephalographic findings that improved during the 18 days stay in SCBU. The baby had a normal neonatal adaptation apart from continuous crying and irritability. Paracetamol was given in an attempt to alleviate pain as the possible cause.

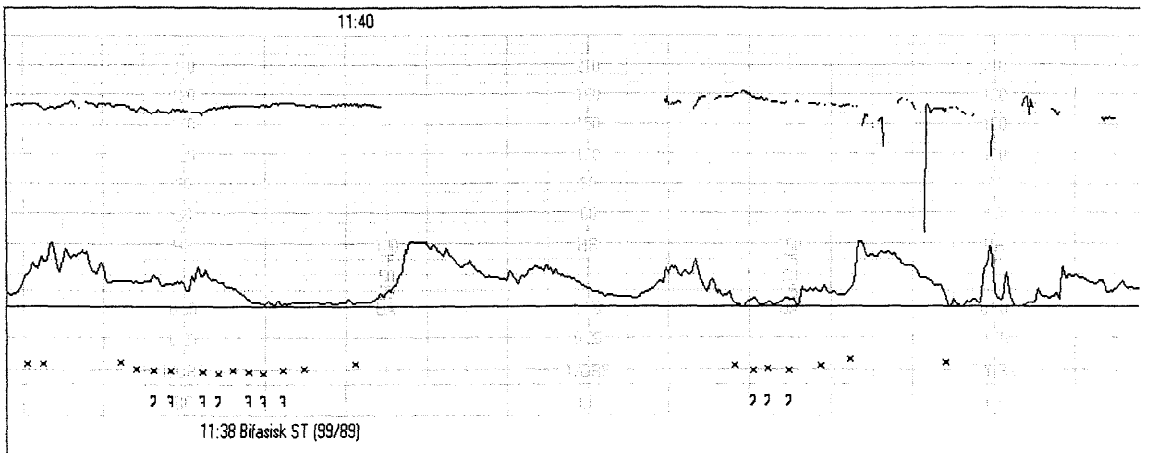
Assessment of the recording
Recording started 08:01 and finished 4 minutes prior to delivery. An abnormal CTG was recorded with decreased variability (silent pattern) and an elevated baseline heart rate to 155 – 160 bpm.
When revealing the ST information, biphasic ST changes were recorded during the last 2 hours of labour.

End of 1st stage recording



End of recording





Comments

The FECG recording lasted four hours and showed a silent pattern with tachycardia. Repeated FBS showed no acidotic values. Intermittent biphasic ST waveforms together with the abnormal CTG trace, indicated abnormality.

It appears as if the fetus was affected prior to the application of a scalp electrode as indicated by a consistently low FHR variability. As uterine contractions became more intense, biphasic ST events emerged as a sign of a decrease in the ability of the myocardium to respond. The cord acid base data is of interest as it indicates a decreased buffering capacity in the cord artery.

Theoretically, in cases where there is a decreased rate of aerobic metabolism in the tissues, the PCO_2 in the fetal blood would tend not to increase and the level of acidosis developing would become less marked. The protons [H^+] generated anaerobically during such circumstances, will be buffered by the tissue buffers and should have less impact on the lowering of blood pH. Thus, an FBS may not indicate acidosis in a situation of a chronic hypoxia with maintained fetal blood flow.

Chronic fetal stress and birth trauma related to a mid cavity ventouse are the probable main causes of neonatal symptoms in this case. A tentorial bleeding and dissection of the carotid artery with embolisation of peripheral arteries and cerebral ischemia are no typical results of intrapartum hypoxia *per see*, but rather of trauma in combination with hypoxia. The CTG+ST guidelines would have indicated operative intervention (CS) 2 hours before delivery took place.

Cases with moderate neonatal encephalopathy in combination with metabolic acidosis or Apgar scores <4 at 5 minutes

Case Lda 0258

Date of delivery: [illegible]

Clinical data
Para 0, normal pregnancy.
Spontaneous onset of labour at 40 weeks of gestation
Clear liquor
Failed mid cavity ventouse followed by cesarean section for non-reassuring fetal heart rate.
No FBS.
Active pushing started 04:00.
Delivery at 04:29

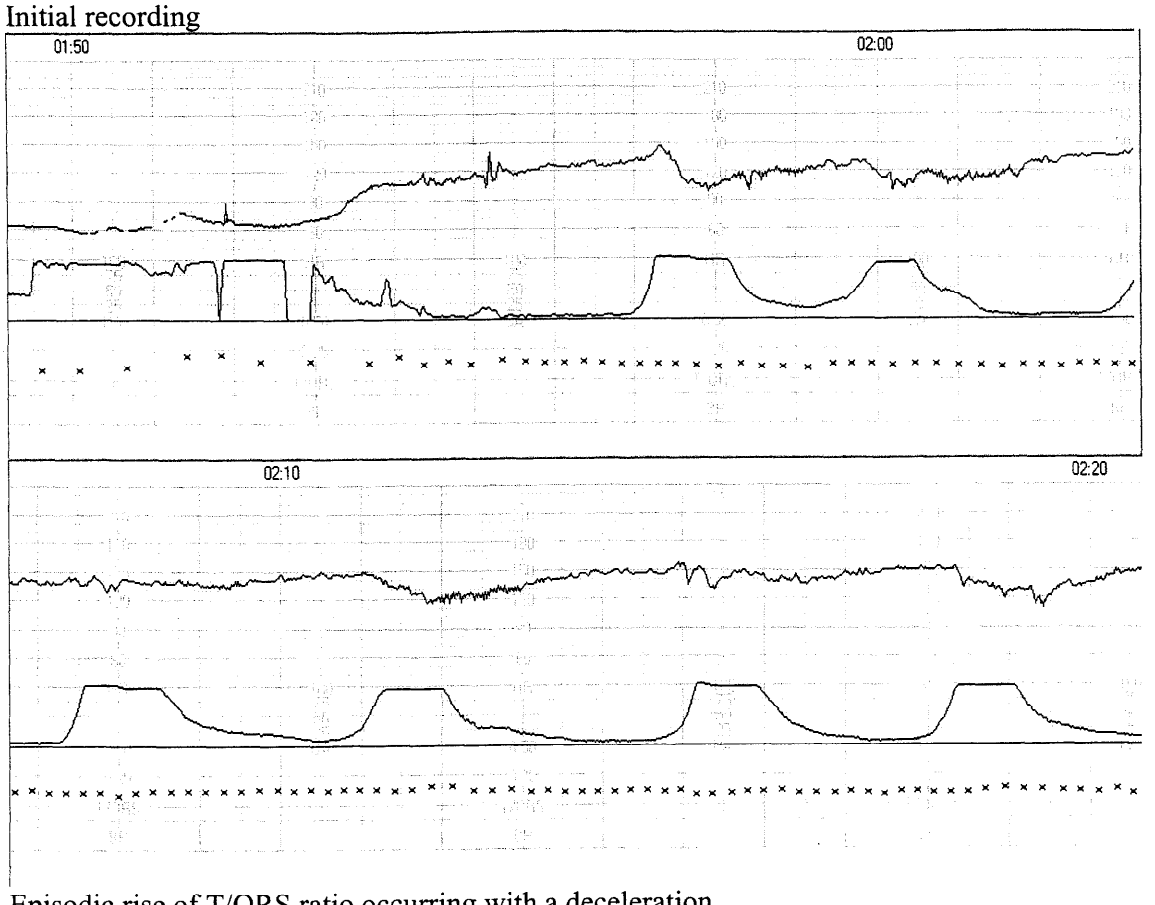
Neonatal data
Female 3900 g
Apgar 2-5-7

Cord artery:	pH 6.78
	PCO_2 18.27 kPa
	BDecf 12.8 mmol/l
Cord vein:	pH 6.84
	PCO_2 15.24 kPa
	BDecf 12.8 mmol/l

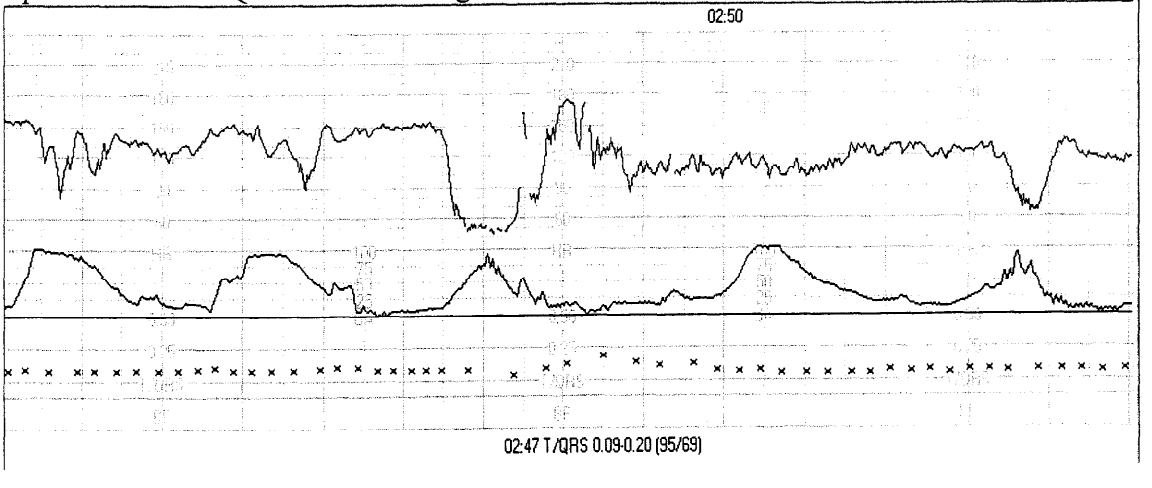
Neonatal outcome
Increase in neuromuscular tone during the first day, there after a normal neonatal period. Nine days in SCBU.

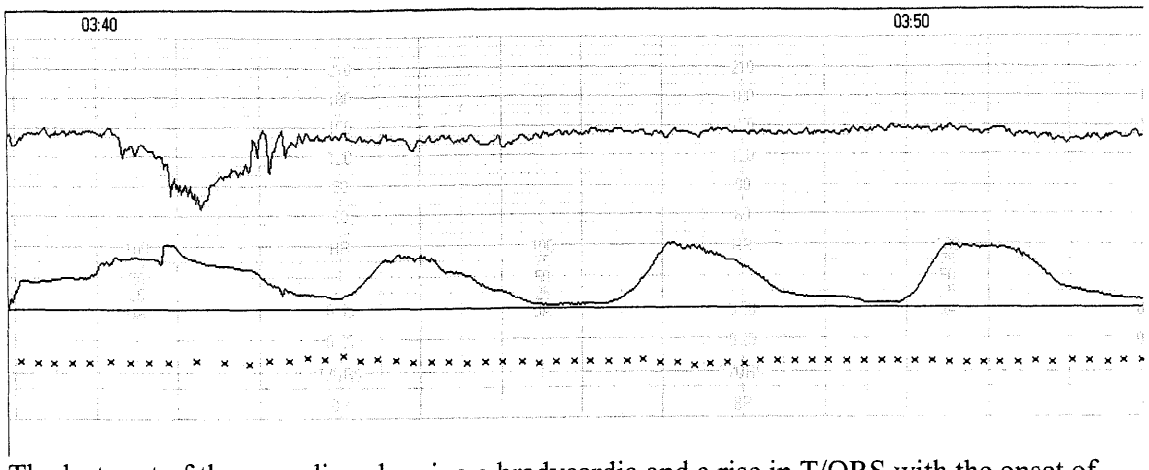
Assessment of the recording
Time 01:50h, at start of recording a marked deceleration with episodic T/QRS rise (not automatically identified by the ST log as <20' of baseline data existed) followed by a gradual increase of baseline FHR to 160 bpm, decreased variability and combined decelerations. At 02:47 another marked deceleration with a significant episodic rise in T/QRS from 0.09 to 0.20, as indicated by the ST-log. The trace showed signs of recovery thereafter. Pushing commenced at 04:00. At 04:08 another deceleration occurred accompanied by a T/QRS rise. The recording was discontinued at 04:10 and delivery by CS occurred at 04:29.

Initial recording

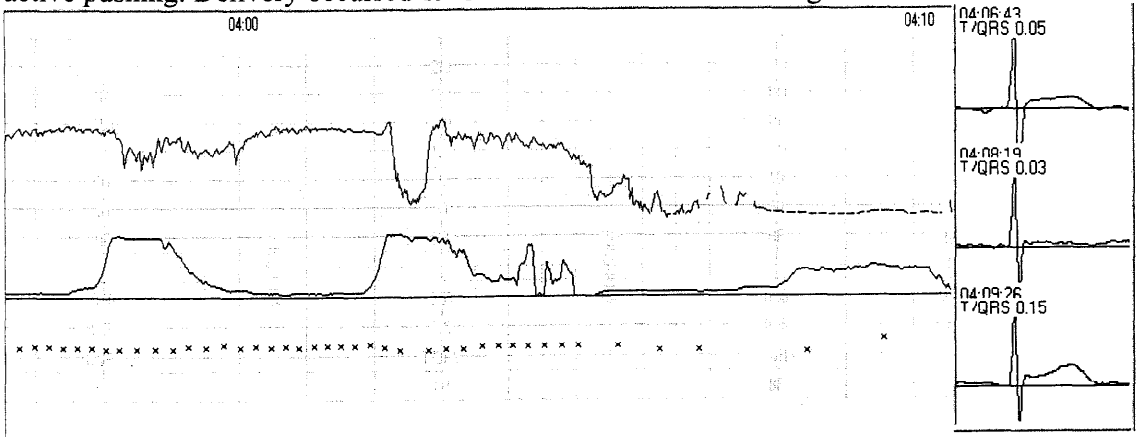


Episodic rise of T/QRS ratio occurring with a deceleration





The last part of the recording showing a bradycardia and a rise in T/QRS with the onset of active pushing. Delivery occurred 19 minutes after end of recording.



Comments:

The fetus showed repeated hypoxic episodes in 1st stage of labour as indicated by decelerations with episodic rise in T/QRS. At the onset of 2nd stage, further signs of a decrease in fetal placental blood flow. A mid cavity ventouse attempt was made, followed by an emergency CS.

The cord acid base indicated metabolic acidosis in the cord artery and vein, supporting the observation of a marked intrapartum hypoxia with neonatal signs of encephalopathy with increased neuromuscular tone.

CTG+ST displayed significant findings at 02:47 indicating an intervention.

Case MAA 479

Date of delivery:

Clinical data
Para 0, gestational age 42weeks + 4 days.
Spontaneous onset of labour.
Meconium stained liquor.
Epidural + oxytocin infusion.
Maternal fever noted and antibiotics given during labour.
Mid cavity ventouse for non-reassuring FHR at 23:30

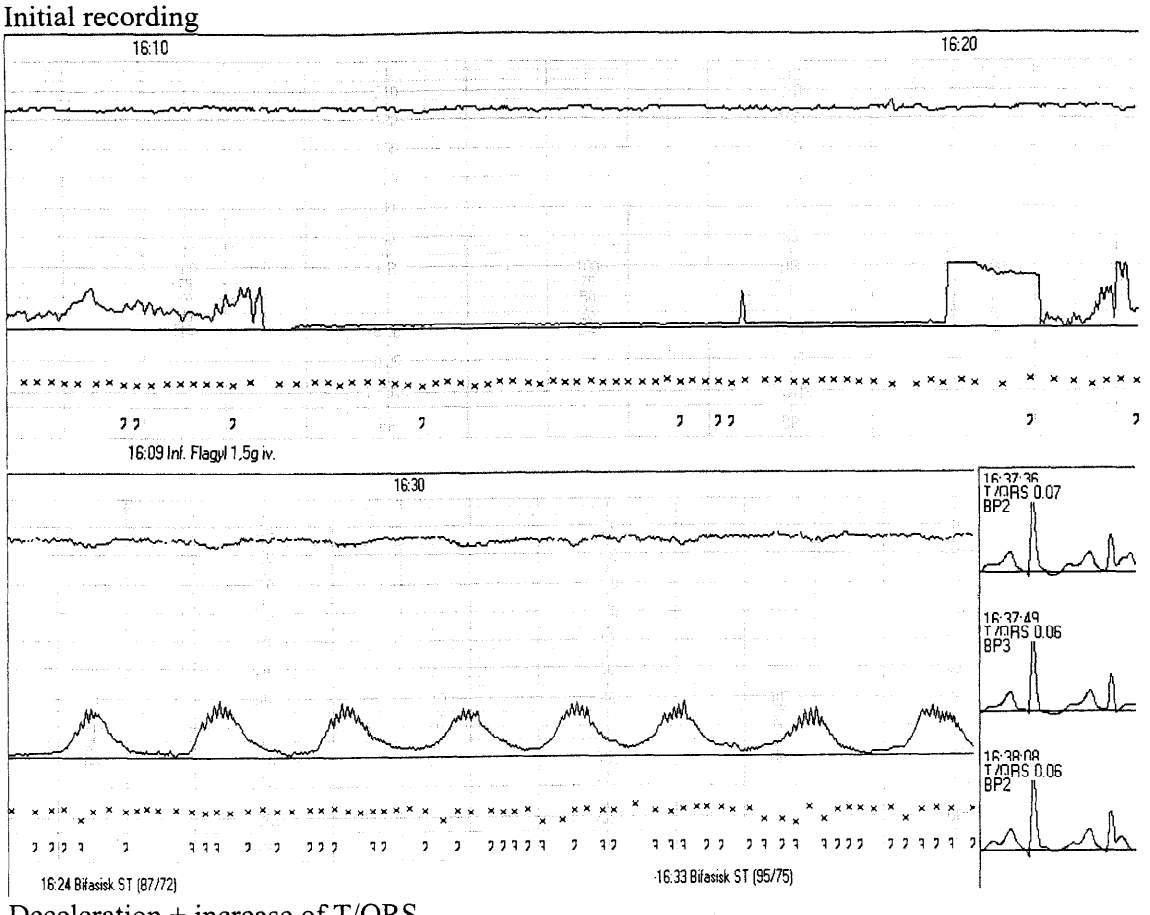
Neonatal data
Female 4160 g
Apgar 1-3-3
Cord artery: pH 6.73
PCO_2 11.38 kPa
BDecf 21.8 mmol/l

Cord vein: pH 6.88
PCO_2 7.76 kPa
BDecf 19.9 mmol/l

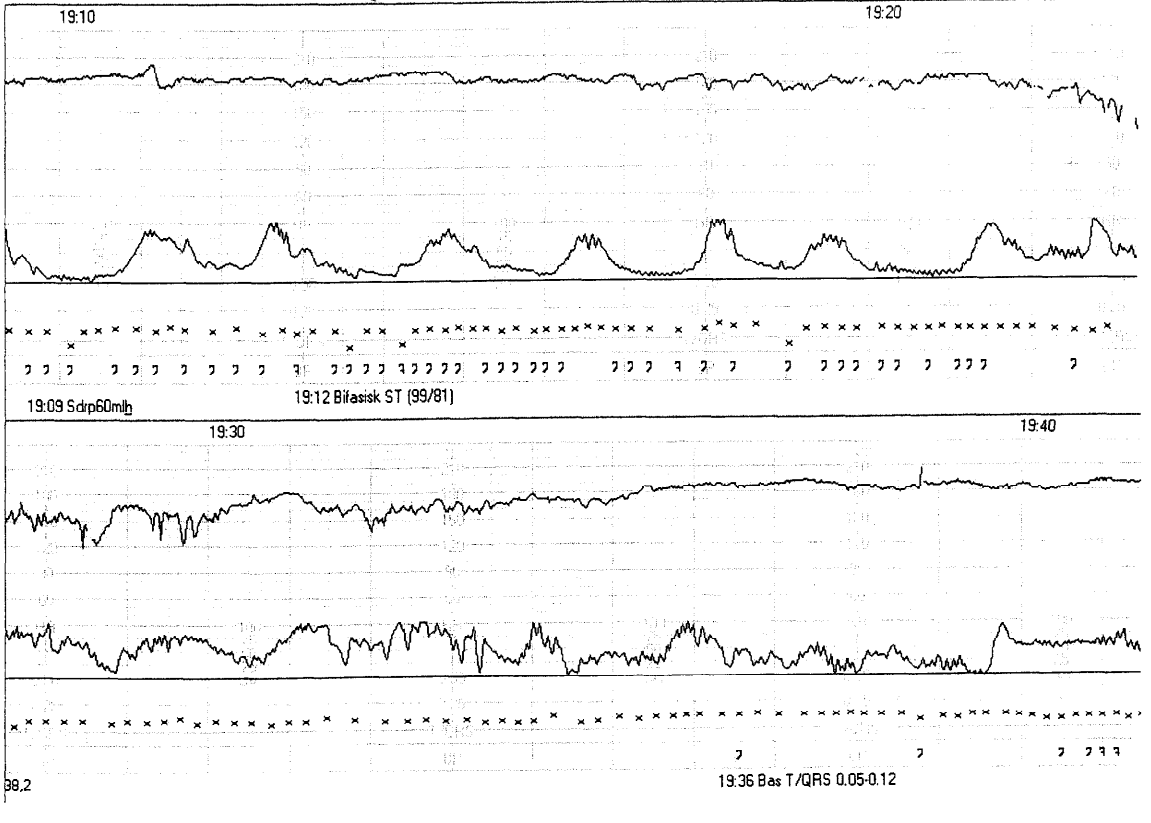
Neonatal outcome
The baby required assisted ventilation with intubation and buffering, spontaneous breathing at 25 min of age.
The baby showed signs of increased neuromuscular tone during the first 24 hours with electroencephalographic abnormalities but without clinical evidence of neonatal seizures.
Elevated serum creatinine (117 mmol/l) during the first 24 hours.
Normal electroencephalogram on Day 2.
Blood glucose in the low range, 5.0 – 3.5 mmol/l.
Treated with phenobarbiturate + antibiotics, C-reactive protein, max 47 mg/l, negative blood cultures.

Assessment of the recording
Recording starts at 13:47 with an increased baseline FHR 170 bpm, at 15:50 further increase to 200 bpm at which time maternal shivering with pyrexia (39.2 ^{0}C). Antibiotics were given intravenously and oxytocin infusion started at 16:20. There was an increased frequency of contractions, 6/10 min, throughout 1st stage of labor. At 16:52, onset of biphasic ST. The pattern with marked tachycardia and continuous biphasic ST continued until 19:28 when a deceleration occurred with more frequent contractions. At 19:30, the ST log identified the first baseline rise of T/QRS and at 20:50 late deceleration started. The recording showed most abnormal CTG patterns with a mix of ST events until end of recording 23:14, sixteen minutes before delivery.

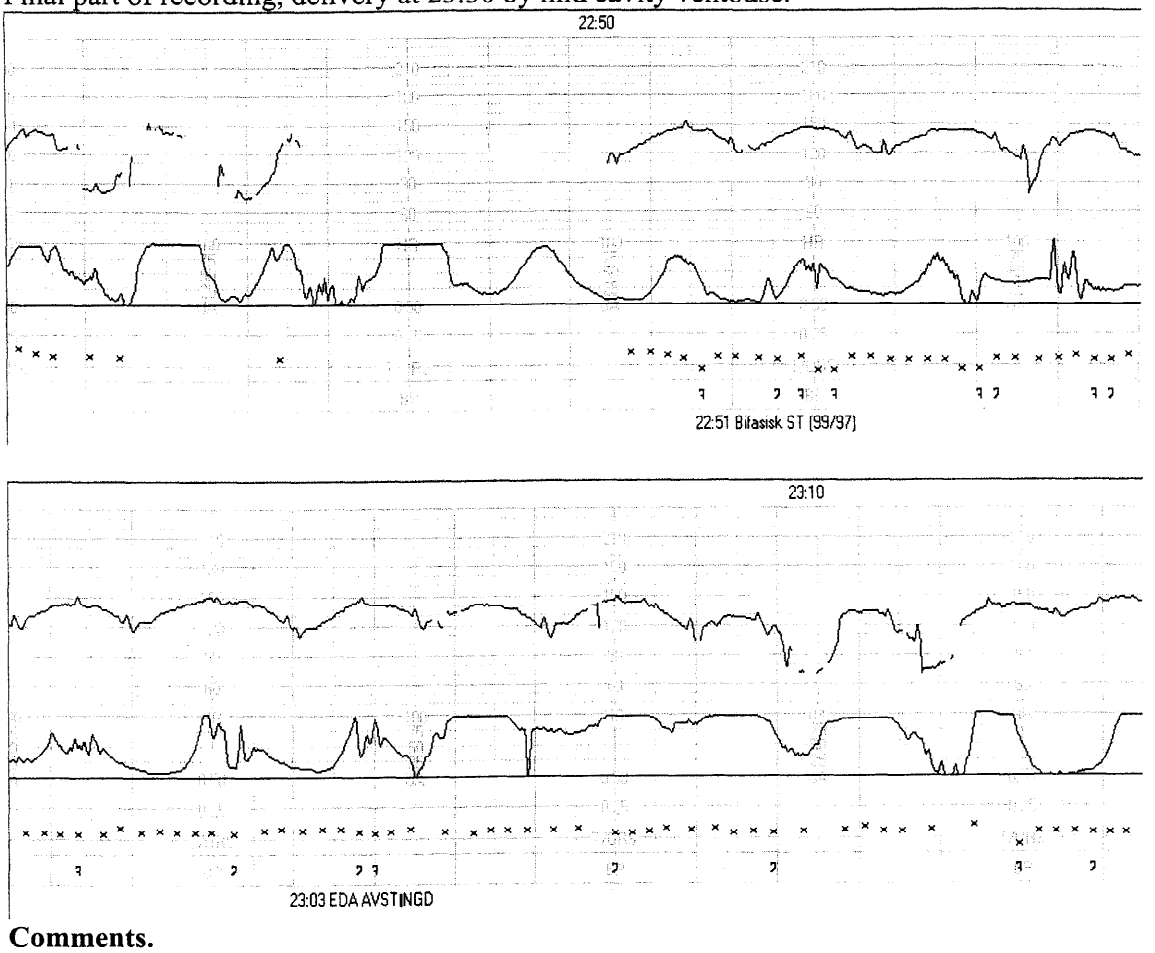
Initial recording



Deceleration + increase of T/QRS



Final part of recording, delivery at 23:30 by mid cavity ventouse.



Comments.

All recorded parameters indicated a fetus affected by the events of labor with maternal pyrexia (infection?) and uterine hyperactivity being causative factors in particular. The initial ST pattern showed biphasic ST associated with tachycardia and increasing uterine activity. It seems possible that a mix of infection related inflammatory agents and hypoxemia *per see* might have affected the fetal myocardium causing such a pattern. As labor progressed, a change in the ST pattern occurred with an increase in T wave height but still ST segment depression, indicating an attempt by the fetal organism to react with an alarm reaction as hypoxia became more severe but still under the influence of inflammatory reactions. Obviously, in a case such as this with the combination of pyrexia and hypoxia, one would expect a high risk of neonatal encephalopathy with signs of multi organ failure. However, the neonate seemed to manage the neonatal period remarkably well with normal behavior and breastfeeding from day 5.

Case MAC 516

Date of delivery: [illegible]

Clinical data
Para 0, normal pregnancy, spontaneous onset of labor at 38 weeks of gestation.
Clear liquor at onset of labor but meconium stained at delivery.
Maternal fever developed after spontaneous rupture of membranes, 15 hours before onset of contractions. Elevated C-reactive protein was noted and antibiotics were given.
Epidural administered at 14:26
Onset active pushing at 15:15.
Normal vaginal delivery at 16:04.

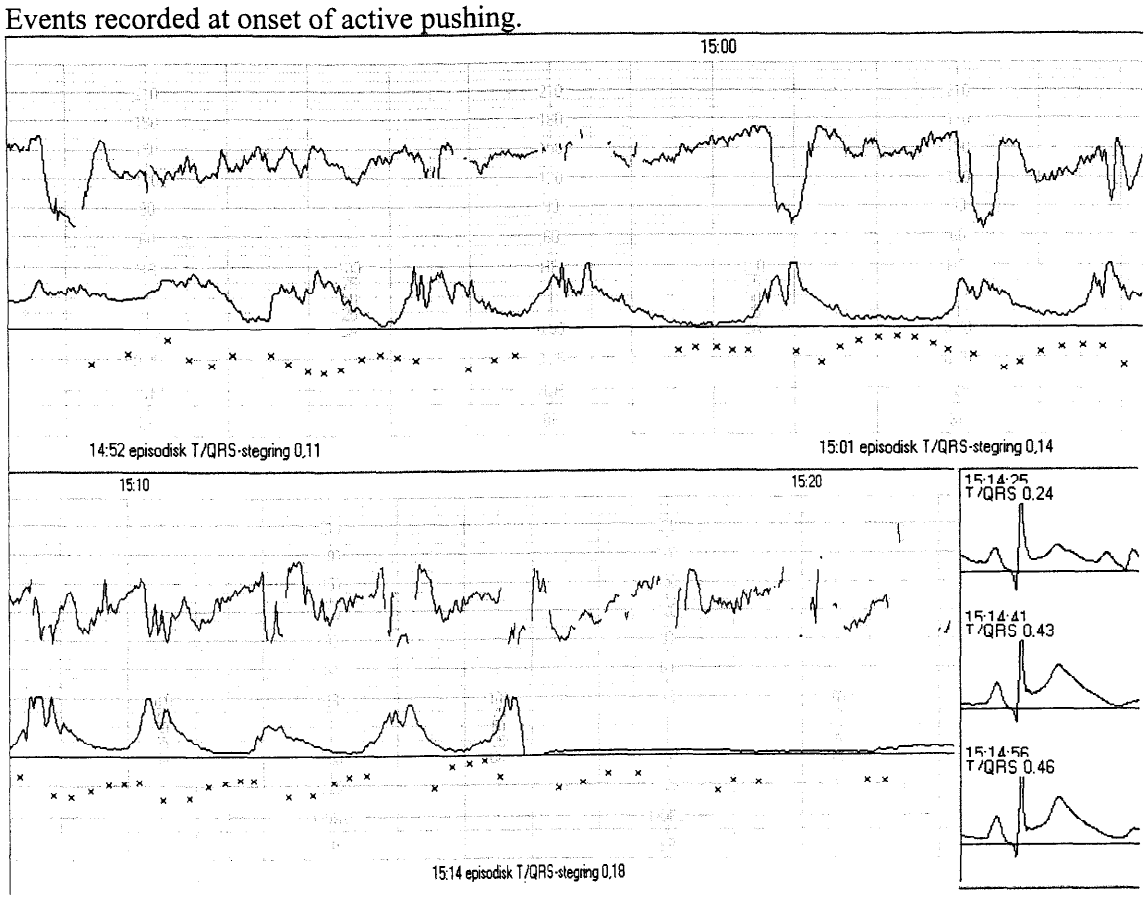
Neonatal data
Male 2705 g (AGA), length 44 cm (-3 SD), craneosynostosis, no chromosomal abnormalities.
Apgar 3-6-7

Cord artery:	pH 6.87
	PCO_2 10.5 kPa
	BDecf 16.9 mmol/l
Cord vein:	pH 6.98
	PCO_2 8.54 kPa
	BDecf 15.1 mmol/l

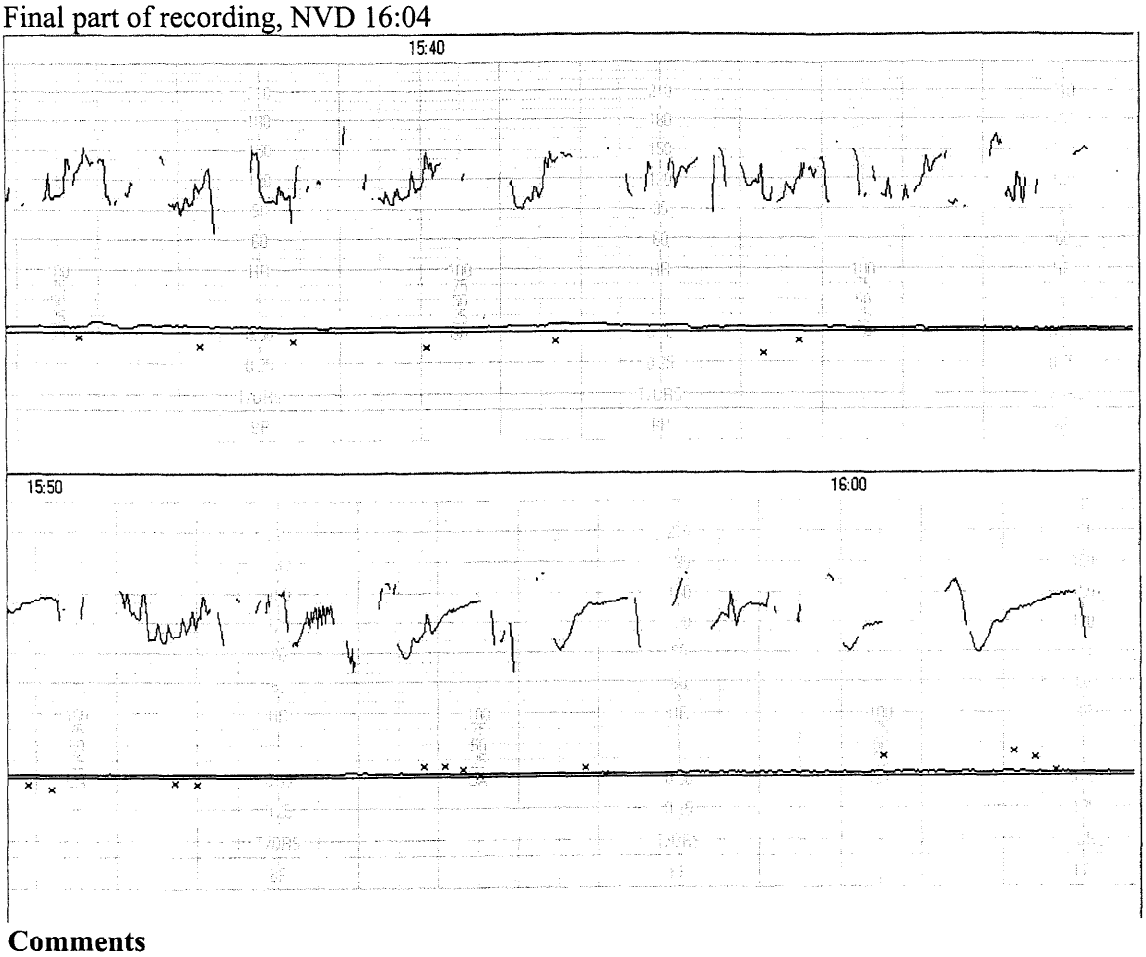
Neonatal outcome
Assisted ventilation with facemask. Treated with CPAP during the first 9 hours for respiratory distress, buffers given. No need for additional oxygen after 18 hrs of age.
Antibiotics given due to an elevated C-reactive protein: 83 mg/l, negative blood cultures.
B-glucose 2.4 mmol/l at 6 hrs of age.
Increased neuromuscular tone but no seizures during the first 24 hrs. CT brain showed normal findings. The baby managed to breastfeed from Day 11.

Assessment of the recording
The recording started at 12:50, showing tachycardia (190 bpm) with intermittent decelerations and normal variability. Baseline HR returned towards 150-160 bpm at 14:50 but decelerations (variable) continued, some of them complicated, until delivery. Poor TOCO signal but signs of increased frequency of contractions.
ST showed abnormalities associated with decelerations throughout the recording, frequent significant episodic and baseline rise in T/QRS indicated by ST-log from 13:05 onwards. A marked rise in T/QRS was recorded at 15:25. A peak T/QRS ratio of 0.73 was noted immediately prior to delivery at 16:04.

Events recorded at onset of active pushing.



Final part of recording, NVD 16:04



Comments

Uncomplicated pregnancy becoming complicated with onset of labor due to maternal pyrexia and signs of bacterial infection after rupture of membranes. Signs of intrapartum hypoxia with onset of active pushing and frequent uterine contractions. The fetus reacted with a marked alarm reaction as indicated by the marked rise in T/QRS with anaerobic metabolism shown by both arterial and venous cord metabolic acidosis. As a consequence of the intrapartum hypoxia, the neonate showed signs of and neonatal distress with respiratory symptoms, hypoglycemia and increased neuromuscular tone.

The baby was short for gestational age but not SGA. Maternal fever and intrauterine infection are possible causes to a reduced ability of the fetus to manage the strain of labor.

The ST log identified abnormalities approximately 2 hours before delivery.

Case MAD 408

Date of delivery: [illegible]

Clinical data

Para 1, normal pregnancy apart from signs of decreased fetal movements and suspected growth retardation (decreased rate of increase in symphys-fundal height), spontaneous onset of labour after 39 weeks of gestation.
Oligohydramnios.
Emergency CS at 17:43 for fetal distress.

Neonatal data

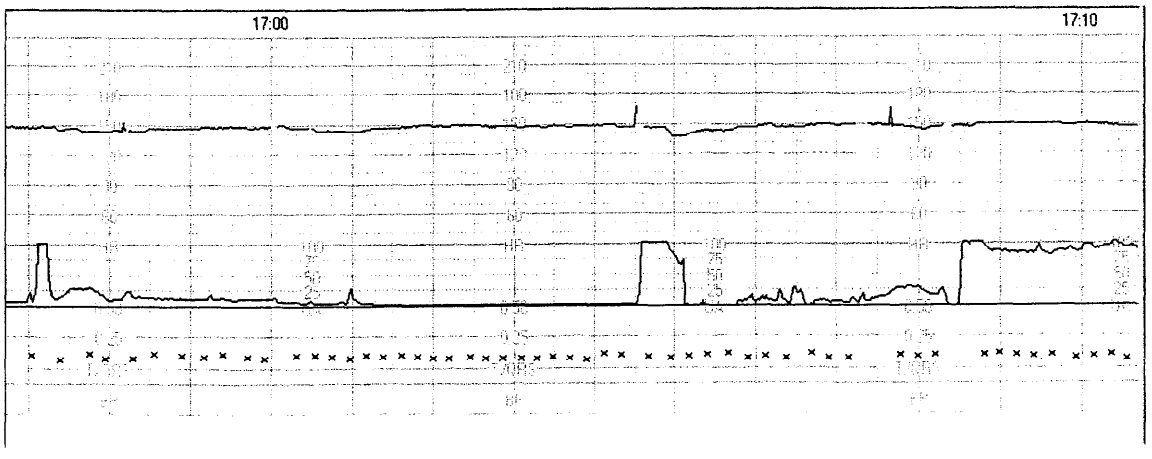
Female 2900 g (previous child with a birth weight of 4000g).
Apgar 1-3-5
Cord artery data not obtained
Cord vein: pH 7.29
PCO_2 6.5 kPa
BDecf 2.5 mmol/l

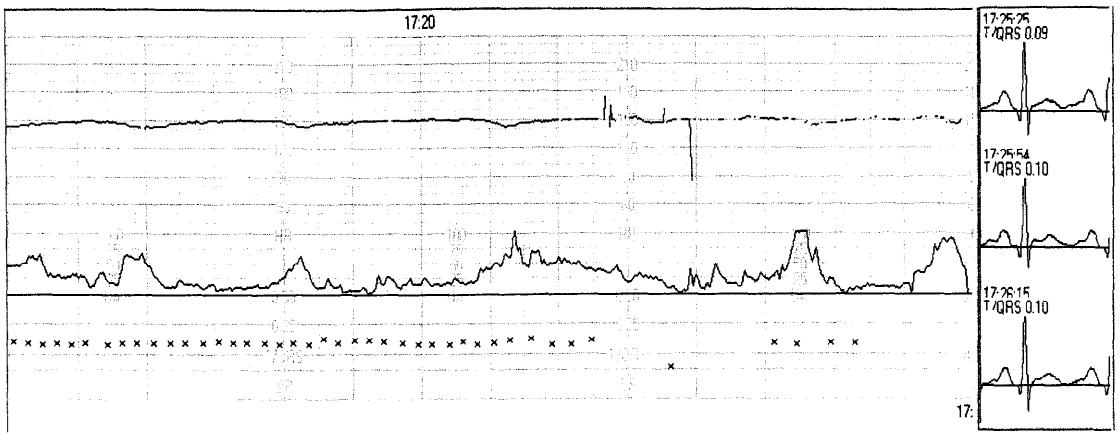
Neonatal outcome

Initial assisted ventilation by facemask followed by intubation, meconium in upper airways.
Adequate breathing at 15 min of age.
Marked hypoglycemia with a blood glucose of 0.5 mmol/l initially.
No sign of meconium aspiration or RDS.
Increased neuromuscular tone but normal electroencephalogram.
No suctioning reflex initially.
Normal behavior after 4 days. Discharged home after 15 days.

Assessment of the recording

Recording started at 16:56 and finished at 17:28.
32 minutes of recording in first stage of labour with preterminal CTG (silent pattern, baseline FHR 150 bpm, shallow decelerations and absent accelerations). No ST events indicated.





Comments

A fetus with signs of reduced placental function as indicated by reduced growth rate, reduced fetal movements, oligohydramnios and preterminal CTG during 32 min of recording. An most unusual case that showed evidence of an alternative fetal adaptation to reduction in placental supply. In this case there is evidence of deterioration starting prior to labor and when the recording started, the fetus had already utilized it metabolic defense and showed signs of a so called hibernating response pattern including loss of fine tuning of the cardiovascular system with no beat-to beat fetal heart rate changes or reactivity and lack of glucose reserves (marked hypoglycemia and lack of metabolic acidosis). No signs of an increase in ST amplitude (anaerobic myocardial metabolism), nor any signs of biphasic ST events (infection, persistent endocardial tissue damage). However, the fetus appeared successful in its choice of action and managed the neonatal period reasonably well.

This case illustrates the need to utilize the information about fetal adaptation to the stress of labor provided by the CTG pattern. It is worth noticing that a preterminal CTG pattern does not necessarily include a bradycardia. The lack of continuous adjustments of the fetal heart rate in a case where no sedative drugs have been given would serve as the best parameter to indicate fetal hibernation, a mechanism of adaptation to stress as physiological as the more common alarm reaction.

Cases with mild neonatal encephalopathy

Case LDD 263

Date of delivery:

Clinical data
Para 0, normal pregnancy, spontaneous onset of labour at 41 weeks of gestation
Meconium stained liquor
Start of active pushing at 18:30.
Outlet ventouse for failure to progress, 19:04

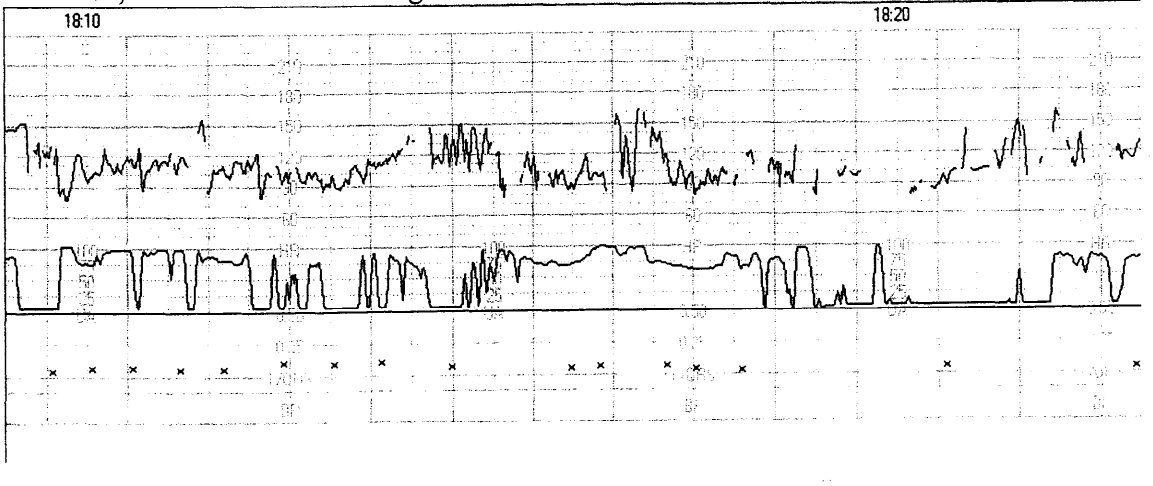
Neonatal data
Male 3530 g
Apgar 2-5-7
Cord artery: pH 7.24
PCO_2 6.36 kPa
BDecf 6.1 mmol/l

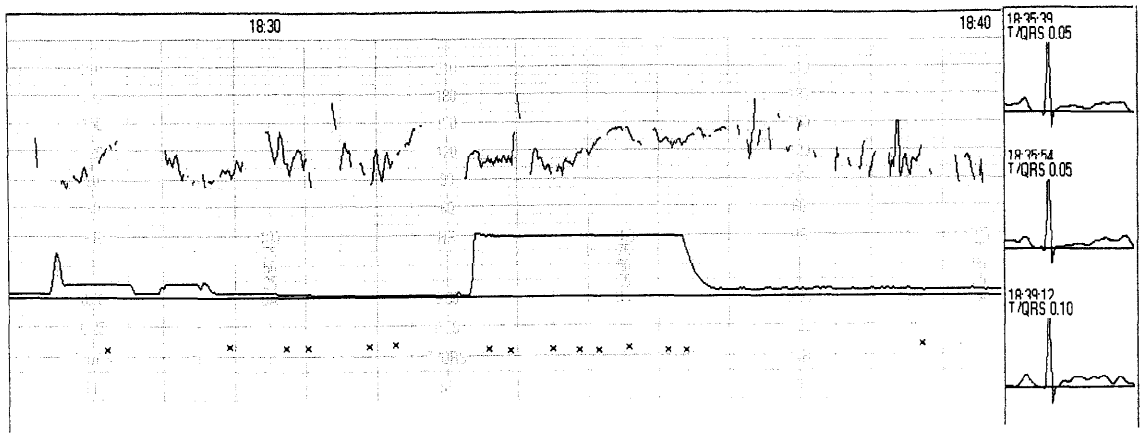
Cord vein: pH 7.27
PCO_2 6.13 kPa
BDecf 5.1 mmol/l
(Blood gases most likely obtained from the same vessel)

Neonatal outcome
Increased irritability during first 24 h, diazepam given
Oliguria treated with diuretics.
On ultrasound examination, signs of intra ventricular hemorrhage grade I.

Assessment of the recording
Recording started 06:40. Perfect recording with normal CTG and ST until the last 20 minutes where there are signs of decelerations from a baseline FHR of 130 bpm with the onset of active pushing. Normal variability. No ST events indicated.

LDD 263, last 30' of the recording





Comment

From the available blood gas data, there is nothing to indicate intrapartum hypoxia. This is in congruence with no ST events indicated by the ST-log. Intraventricular hemorrhage in a term baby without intrapartum hypoxia would favor trauma (outlet ventouse) as a cause.

Cases with Metabolic acidosis + admittance to Special Care Baby Unit

Case OEK 394

Date of delivery: [illegible]

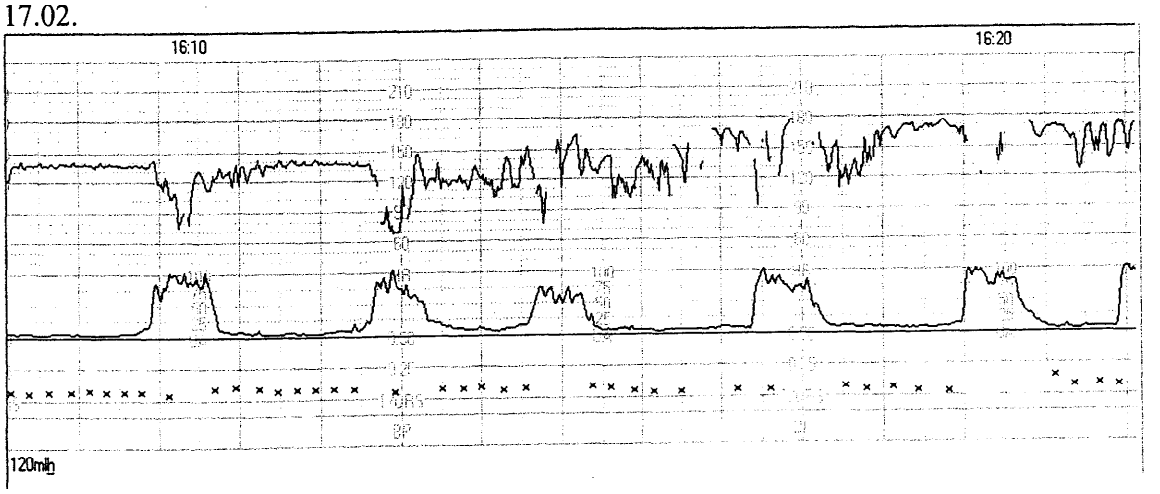
Clinical data
Para 0, normal pregnancy, spontaneous onset of labor after 40 weeks of gestation.
Augmented labor.
Normal vaginal delivery at 17:02

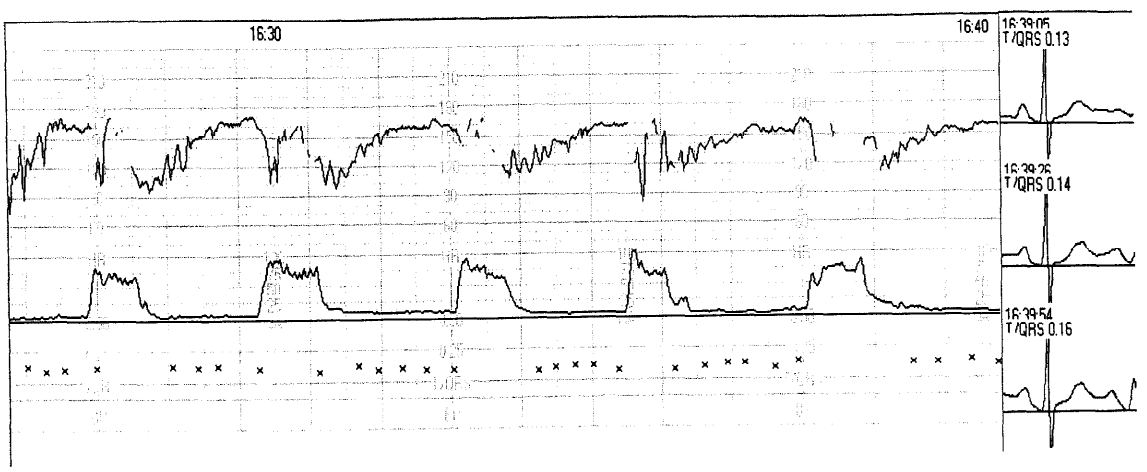
Neonatal data
Male 3115 g
Apgar 6-9-9
Cord artery: pH 7.01
PCO_2 7.06 kPa
BDecf 15.8 mmol/l

Cord vein: pH 7.06
PCO_2 5.94 kPa
BDecf 15.7 mmol/l

Neonatal outcome
Buffer given at 1 hour of age.
Neonatal acid base: Venous pH 7.11, BDecf 12 mmol/l.
Respiratory symptoms with need for extra oxygen during the first 10 hours.
Pneumonia on X-ray, C-reactive protein 72 mg/l.
Hyperbilirubinemia treated with phototherapy.
No hypoglycemia.
SCBU stay: 7 days, uncomplicated neonatal course otherwise.

Assessment of the recording
Normal CTG until 2nd stage. Thereafter an increase of baseline FHR to 160 bpm accompanied by combined late decelerations. Normal variability. After onset of late decelerations, a gradual T/QRS rise is noted over a 30 minute period, from 0.06 - 0.13. Normal vaginal delivery at 17.02.





Comments

Intrapartum hypoxia during 2nd stage as indicated by CTG+ST changes and biochemical findings. A significant ST event occurred 22 min before delivery. Apart from immediate neonatal acidosis and some respiratory symptoms, uneventful neonatal period.

Case OEJ 330

Date of delivery: [illegible]

Clinical data

Para 0, normal pregnancy apart from cholestasis with severe itching causing induction of labor after 38 weeks of gestation.
Clear liquor, epidural
FBS
21:58 - pH 7.31
00:12.- pH 7.26
NVD at 01:31

Neonatal data

Male 3860 g
Apgar 3-7-8
Cord artery: pH 7.04
PCO_2 9.33 kPa
BDecf 10.3 mmol/l

Cord vein: pH 7.08
PCO_2 6.58 kPa
BDecf 13.6 mmol/l

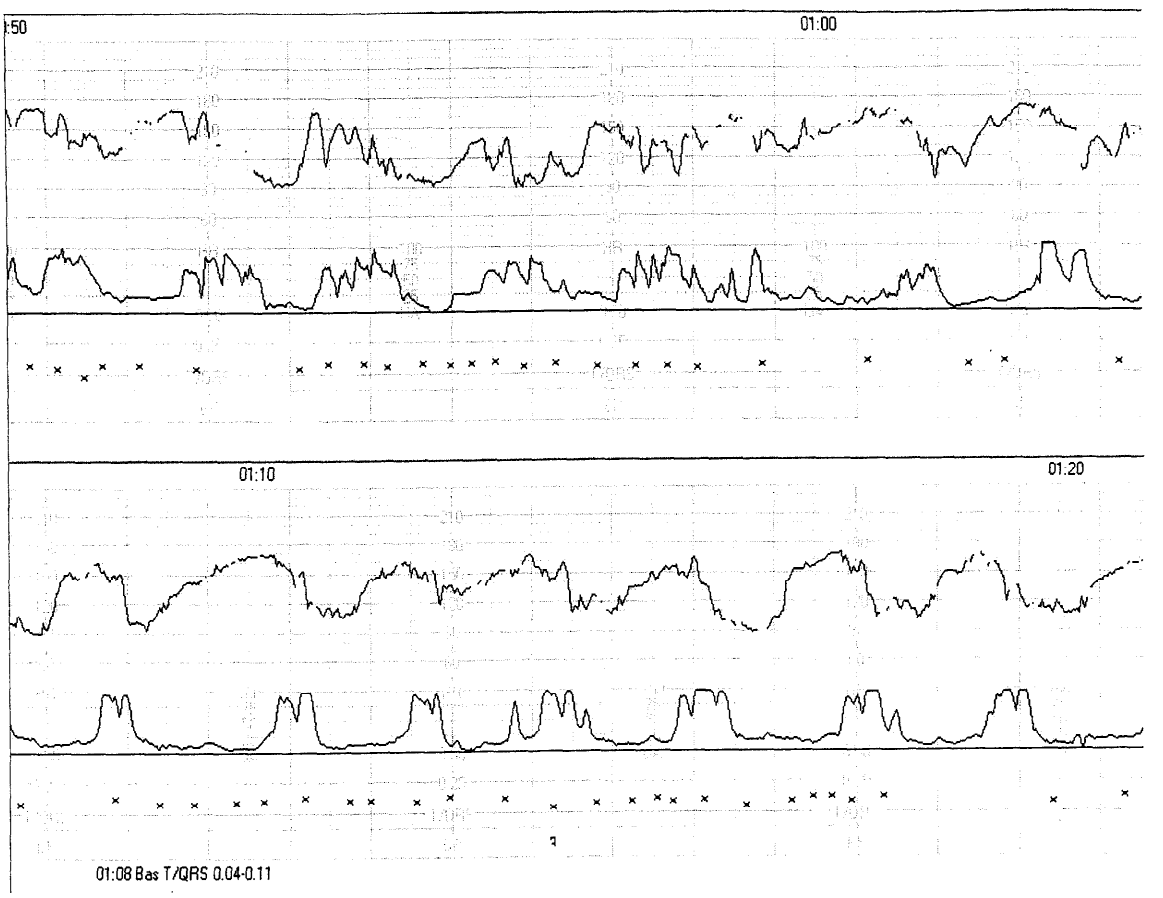
Neonatal outcome

Venous sample obtained at 19 min of age: pH 7.00
PCO_2 7.00 kPa
BDecf 16.3 mmol/l
Lactate 13.2 mmol/l rising to 14.8 mmol/l.
Normal glucose levels (3.4 mmol/l).

Buffer given due to acidosis and respiratory grunting – normal respiratory pattern after buffering. Two uneventful days in SCBU.

Assessment of the recording

CTG with a baseline FHR of approximately 160 during 1st stage, normal reactivity and variability. Progressive CTG changes in 2nd stage with tachycardia, late decelerations and low variability. Abnormal pattern started at 00:50 h, i.e. 41 min before delivery.
ST-log indicated a significant T/QRS baseline rise at 01:08.



Comments

Intrapartum events indicating hypoxia during active pushing in 2nd stage of labor. Neonate moderately affected with cord vein metabolic acidosis, metabolic acidosis immediately after birth, high lactate and a need for buffering. ST event log indicated intervention 33 min before delivery.

Case LDC 364

Date of delivery: [illegible]

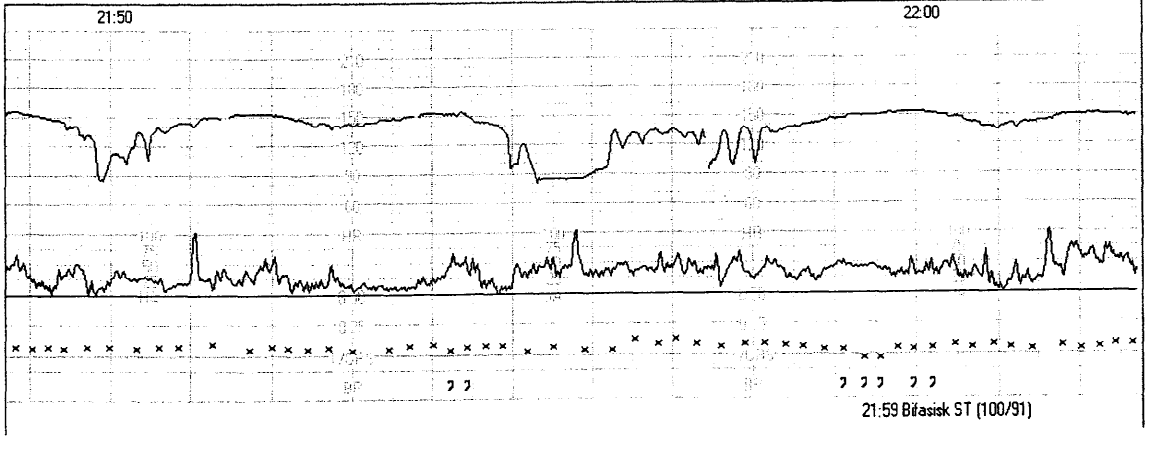
Clinical data
Para 0, norm pregnancy, spontaneous onset of labor after 40 weeks of gestation.
Meconium stained liquor.
Epidural
FBS 23:00 - pH 7.03
Emergency CS at 23:27

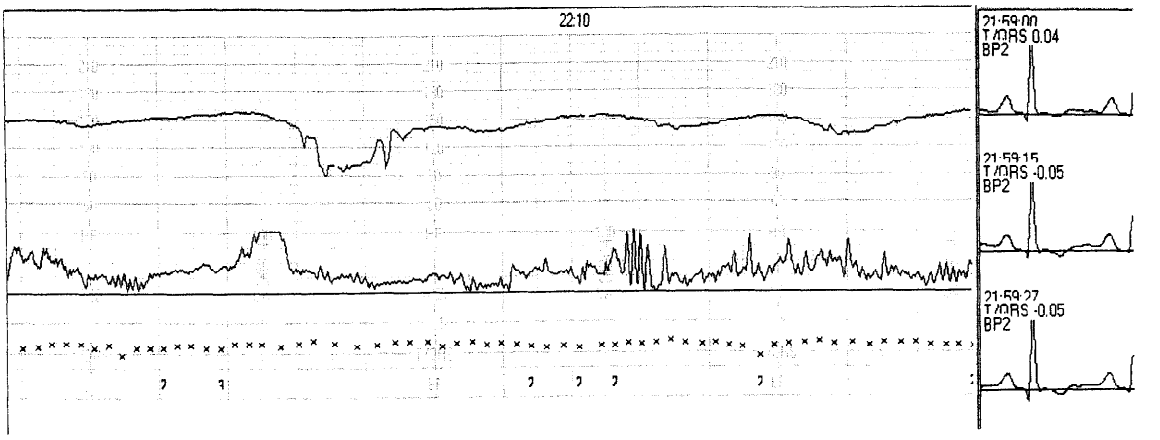
Neonatal data
Female 3739 g
Apgar 1-6-10
Cord artery: pH 7.01
PCO_2 7.69 kPa
BDecf 14.7 mmol/l

Cord vein: pH 7.08
PCO_2 7.07 kPa
BDecf 12.6 mmol/l

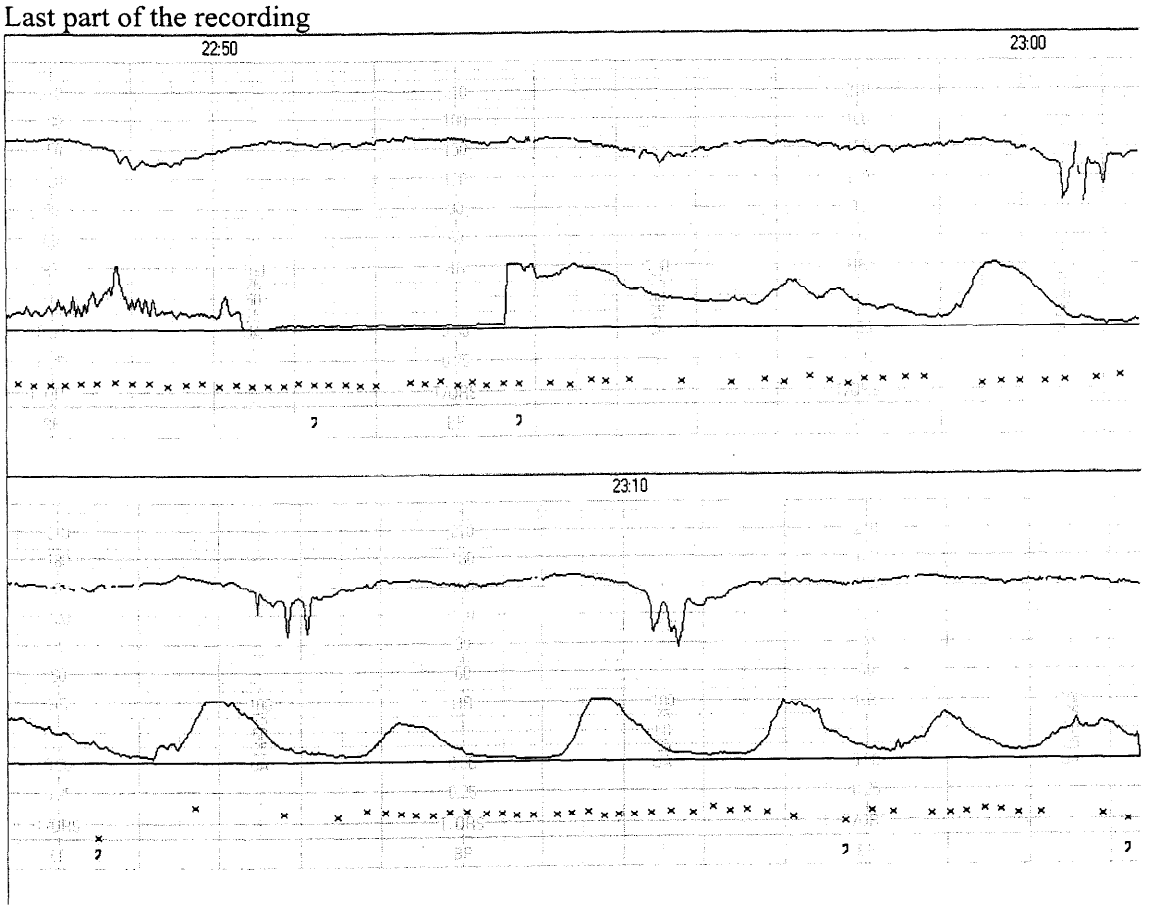
Neonatal outcome
Baby initially tired but no neuromuscular abnormalities, 6 days in SCBU.

Assessment of the recording
At onset of recording at 20:13, the CTG showed a baseline FHR of 130bpm, late decelerations and normal variability. At 21:40, a most abnormal CTG developed with loss of variability, increased baseline FHR to 160 bpm and late decelerations. A biphasic ST event was indicated at 21:59.





Last part of the recording



Comments

Signs of CTG changes already at onset of recording becoming more abnormal as labour continued showing a preterminal pattern with no or very little reactivity. Still this CTG pattern required to be confirmed by an FBS before intervention by emergency CS was undertaken. Note the delay of 27 minutes from obtaining the FBS to delivery of a case of combined cord artery and vein metabolic acidosis. Biphasic ST indicated 1.5 h before delivery a myocardium unable to respond.

Case LDA 275

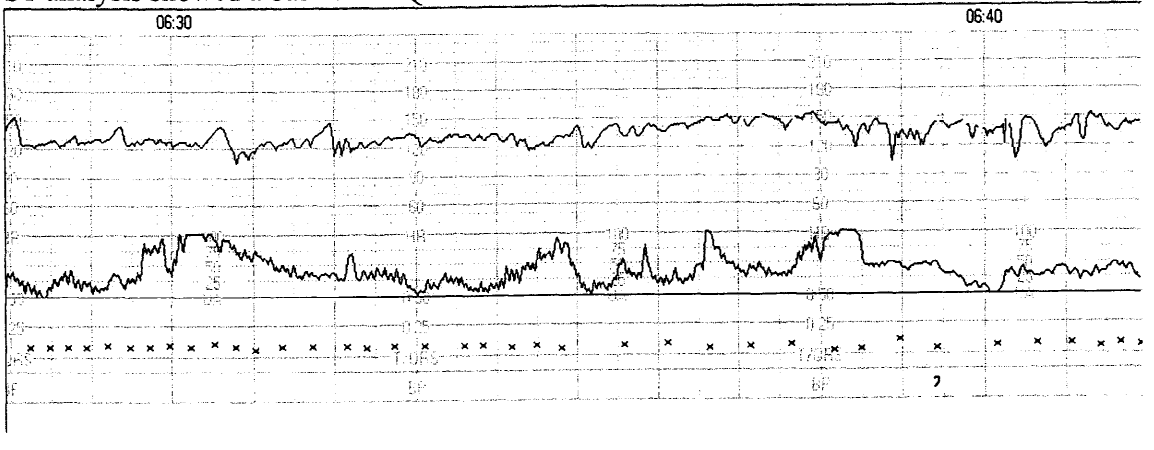
Date of delivery: ~~~

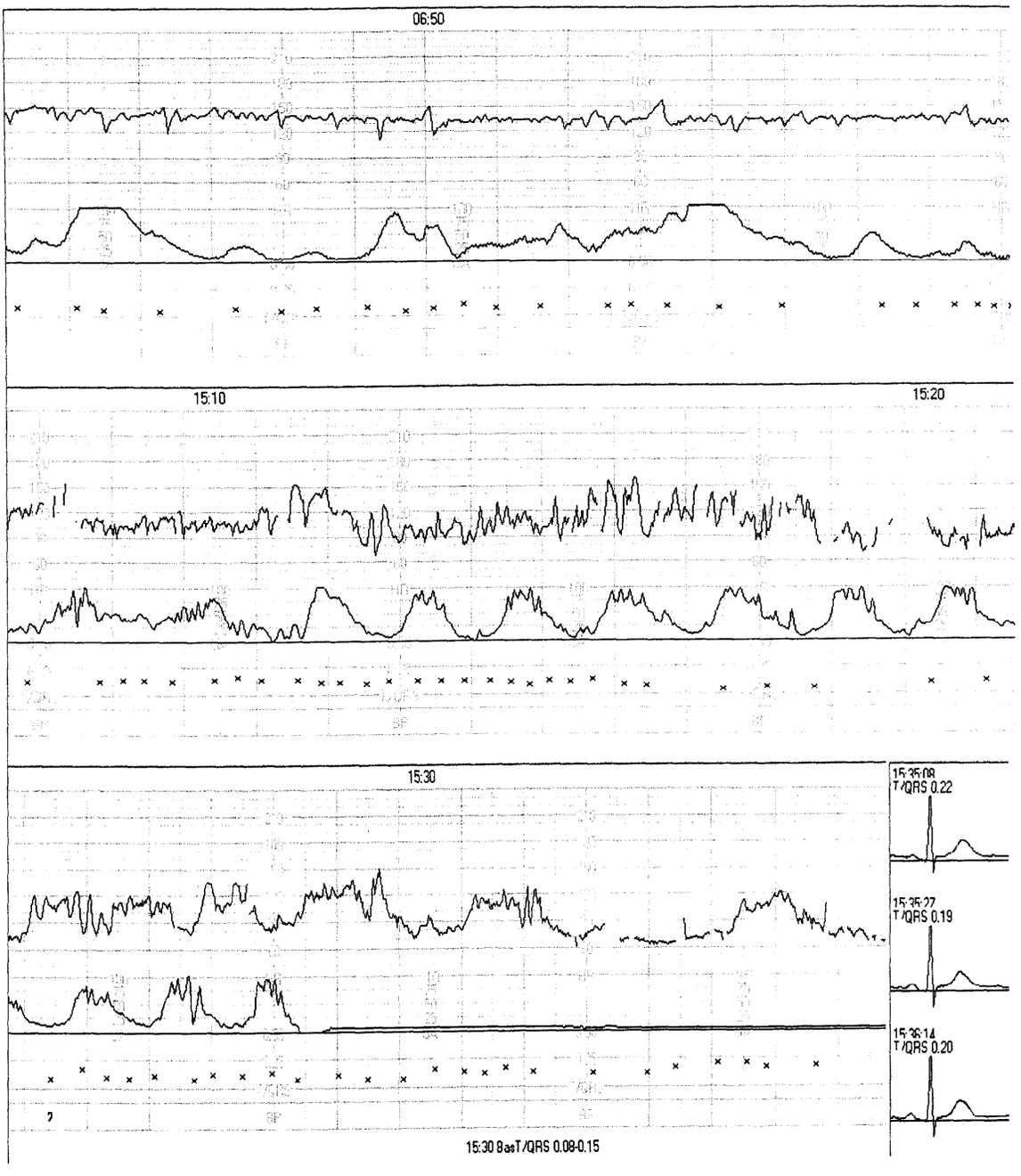
Clinical data
Para 0, spontaneous onset of labor after 40 weeks of gestation
Clear liquor,
Epidural + Pethidine analgesia.
Onset active pushing: 15:20
Mid cavity ventouse for fetal distress at 15:45.

Neonatal data
Female 3970 g
Apgar 1-8-9
Cord artery: pH 7.04
PCO_2 8.35 kPa
BDecf 12.1 mmol/l

Neonatal outcome
Initial respiratory symptoms with tachypnea, requiring extra oxygen during the first hours, thereafter an uncomplicated neonatal period.

Assessment of the recording
Normal CTG pattern until onset of active pushing in 2nd stage of labor. At 15:00, onset of decelerations with periods of increased heart rate variability followed by complicated decelerations.
ST analysis showed a baseline T/QRS rise at 15:30.





Comments

Acute hypoxia emerging at onset of active pushing. Simultaneously occurring changes in fetal heart rate and ST. The latter identifying an active fetal response with an alarm reaction, moderate metabolic acidosis at delivery and short lasting neonatal symptoms related to some delay in the cardio-pulmonary adaptation process.

Case LDA 298

Date of delivery:

Clinical data
Para 0, spontaneous onset at 40 weeks of gestation, normal pregnancy.
Onset active pushing: 14:30.
Failed mid cavity ventouse for FTP followed by forceps, 15:05

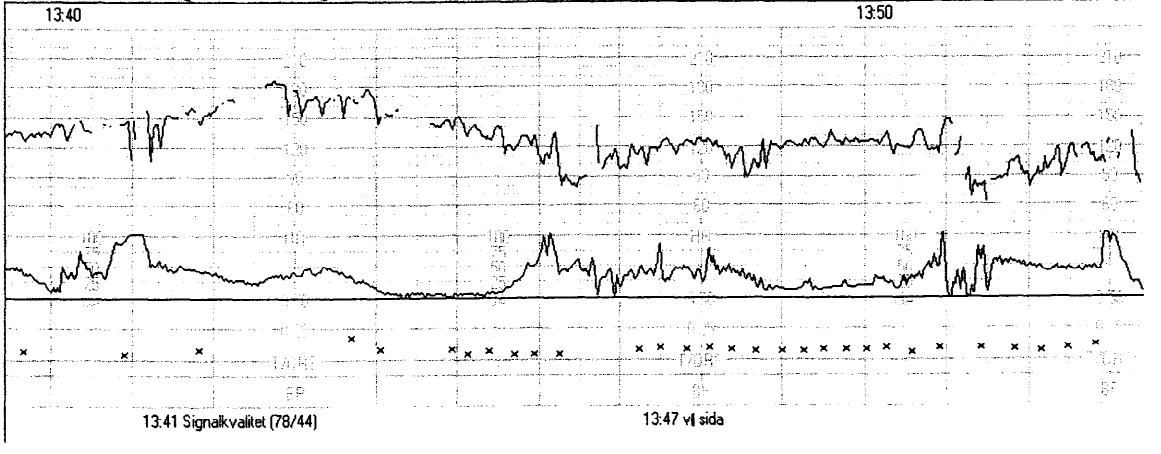
Neonatal data
Male 2815 g
Apgar 7-8-8
Cord artery: pH 7.03
PCO_2 7.70 kPa
BDecf 13.7 mmol/l

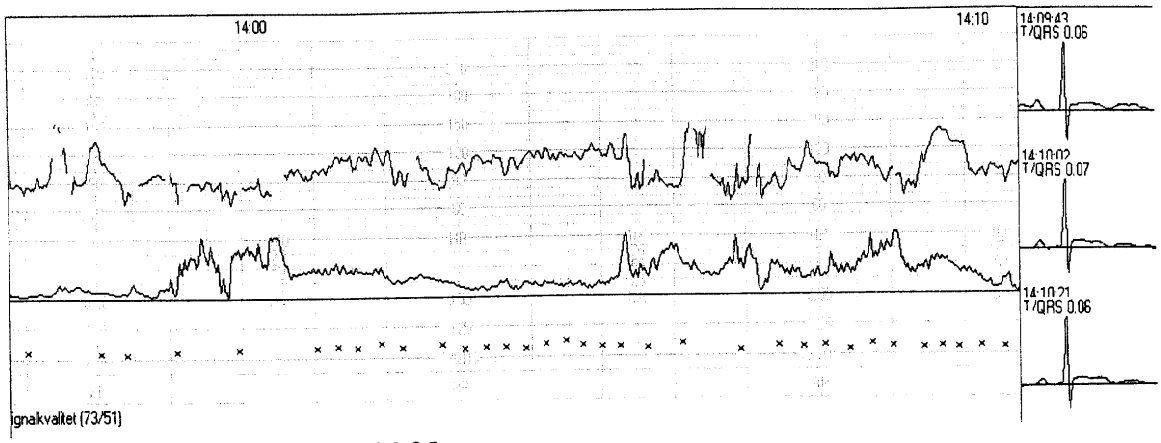
Cord vein: pH 7.05
PCO_2 6.87
BDecf 14.4

Neonatal outcome
Uneventful 2 days visit in SCBU.

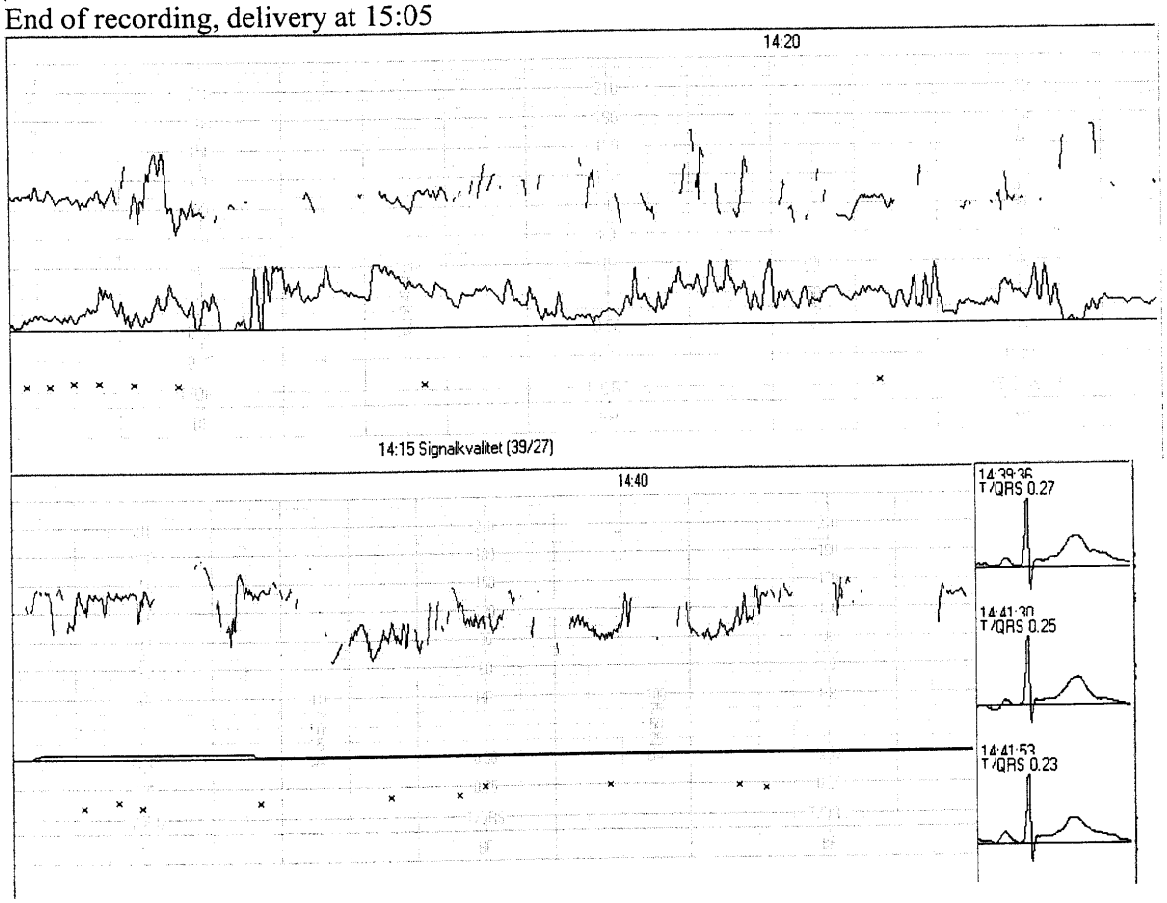
Assessment of the recording
Normal CTG with baseline FHR 110 bpm, normal variability and reactivity until end 1st stage. At this point in time some episodic increase in FHR occurred followed by decelerations with maintained variability.
ST with loss of data prohibiting automatic ST assessment but visual analysis shows a baseline T/QRS rise from approx. 14:30.

End of first stage recording





End of recording, delivery at 15:05



Comments.
Hypoxia developing during active pushing in 2nd stage of labour. The rise in T/QRS identifying an active fetal response with an alarm reaction, moderate metabolic acidosis at delivery and a neonate managing the situation well.

Case OEF 305

Date of delivery: [illegible]

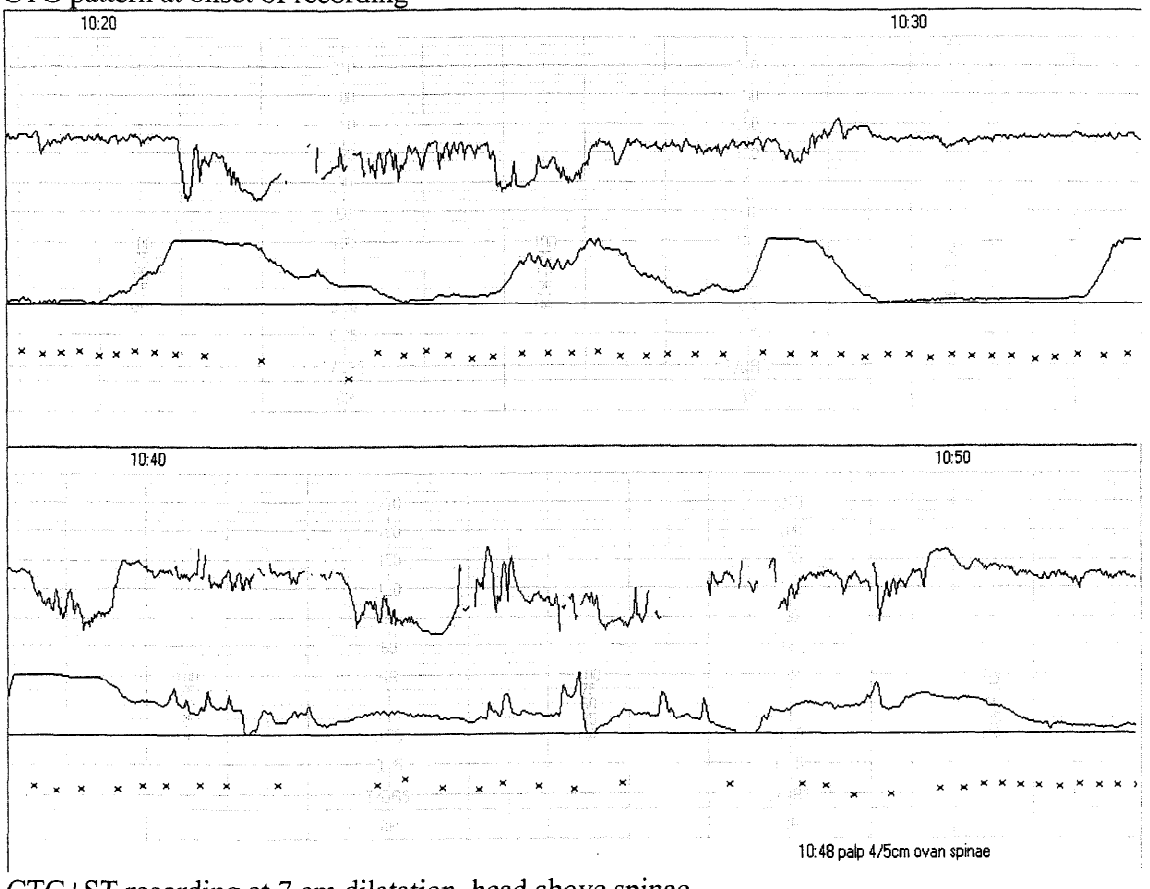
Clinical data
Para 0, maternal epilepsia, Phenantoin treatment.
Normal pregnancy, Spontaneous labour after 42+2 weeks of gestation
Clear liquor.
Epidural at 11:25.
FBS 1 [illegible] at 23:15 pH 7.28 (normal)
FBS 2 [illegible] at 01:06 pH 7.08 (acidotic)
Active pushing: at 00:50
Uncomplicated mid cavity ventouse at 01:27 due to a low scalp pH, thick meconium at time of delivery.

Neonatal data
Female 4040 g
Apgar 2-3-7
No cord data due to cord rupture at time of delivery. Cord around the neck.

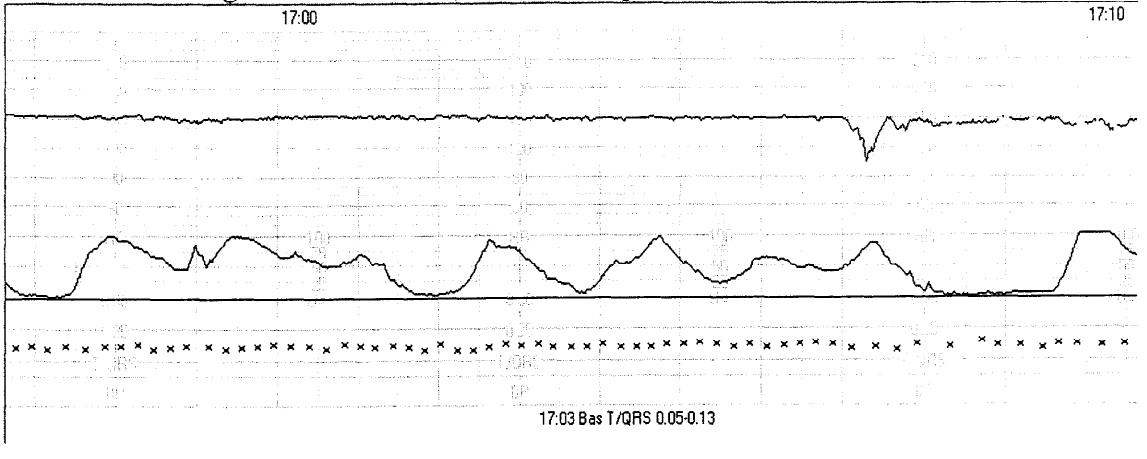
Neonatal outcome
Active resuscitation with ventilation by facemask and suctioning of upper airways, responded with heart rate >100 bpm but delay in onset of breathing until 4-5 minutes, first cry at 6.5 min of age.
To neonatal ward and buffer solution given.
No respiratory symptoms, but some problems with breastfeeding, good suctioning reflex. Slight signs of increased neuromuscular tone noted at 24 hrs of age, no seizures, oliguria during the first 2 days. Laboratory findings revealed increased creatinine, urea and liver enzymes during the first 3 days post partum.
Electroencephalographic recording without clear abnormalities.

Assessment of the recording
Good quality recording lasting 15 hrs, finishing at 01:14, i.e. 13 min before delivery. According to the notes, the CTG recording called for concern already at onset. Oxytocine infusion started at 13:20. At 13:32, meconium stained liquor was noted. Early in 1st stage from 16:30 to 18:30 a progressive increase of T/QRS by 0.09 associated with loss of variability and baseline FHR increasing to 155 bpm, a 7 min episode of bradycardia at 18:16. After that a pattern of tachycardia and deep early and combined decelerations occurred, with maintained variability. Increased uterine activity during these hours of recording.

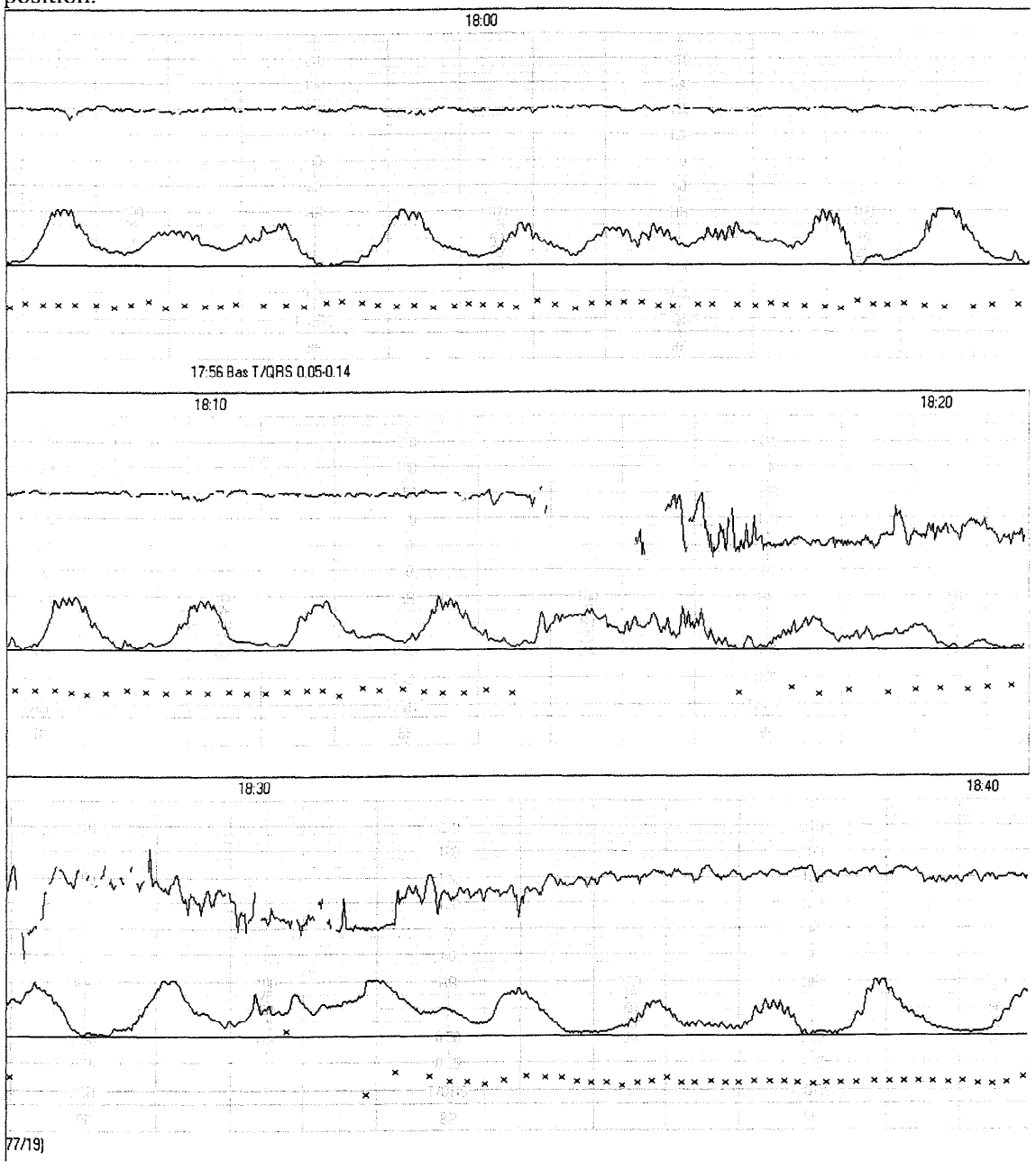
CTG pattern at onset of recording



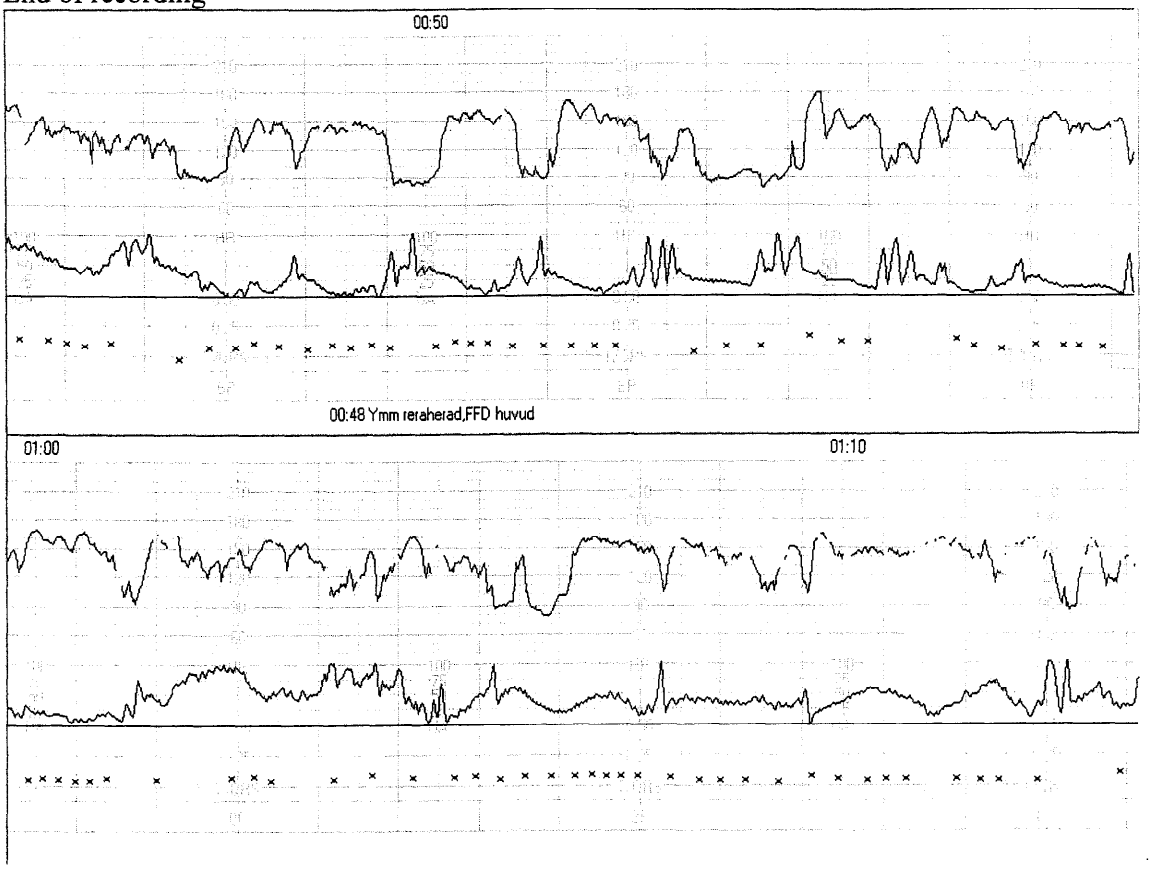
CTG+ST recording at 7 cm dilatation, head above spinae.



Epidural top-up with Marcain at 18:15, deceleration occurred in association with change of position.



End of recording



Comments.
An interesting case delivered post term with uterine hyper stimulation and known cord entanglement, meconium during delivery, need for active resuscitation and signs of hypoxic multi-organ involvement but no signs of neonatal encephalopathy.
Significant CTG+ST events were recorded already in 1st stage of labour. No FBS was obtained at this point in time. When an FBS was obtained, the situation had improved. as the fetus seemed to recover with return of heart rate variability in association with an arousal reaction related and improved cord blood flow?.
Intervention done because of a low scalp pH obtained in 2nd stage.
Neonatal multi-organ symptoms indicated a more long-lasting hypoxic process. Revealing the ST event log would have alerted the physician to intervene already at 17:03 (pathological CTG and significant T/QRS rise), i.e. almost 7.5 h before the actual delivery.

Case OEB 363

Date of delivery: [illegible]

Clinical data
Para 0, normal pregnancy, spontaneous labour after 40 completed weeks of gestation
Meconium stained liquor
Active pushing commenced at 01:50
Outlet ventouse for threatening asphyxia at 03:14

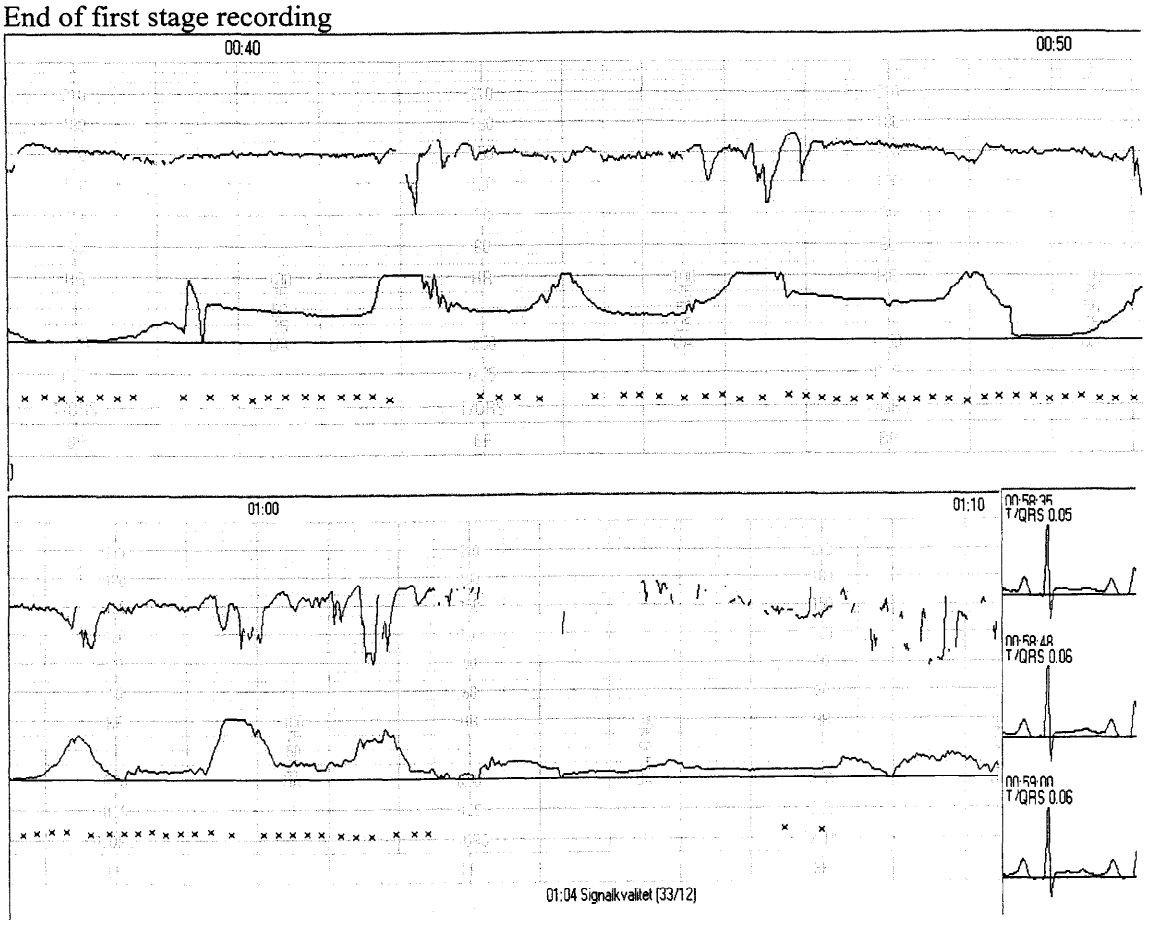
Neonatal data
Male 2465 g (SGA)
Apgar 5-8-8

Cord artery:	pH 6.80
	PCO_2 11.91 kPa
	BDecf 18.4 mmol/l
Cord vein:	pH 6.93
	PCO_2 8.33 kPa
	BDecf 17.2 mmol/l

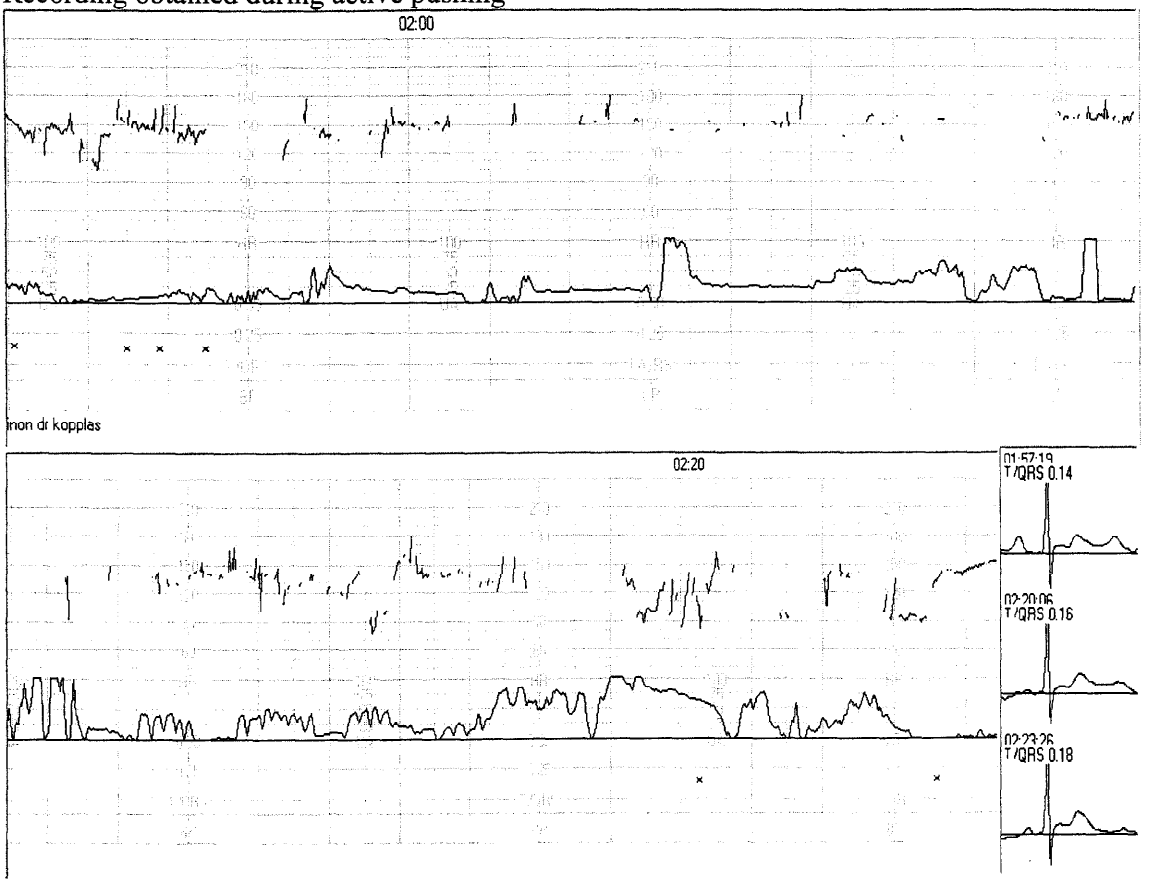
Neonatal outcome
Suctioning of thick meconium before first breath. Spontaneous ventilation at 4 min, buffers given. Hypoglycemia with blood glucose of 1.1 mmol/l at 3.5 hrs of age. Meconium aspiration syndrome requiring continuous positive airway pressure (CPAP) and extra oxygen the first two days. No neuromuscular abnormalities. Hyperbilirubinemia requiring light therapy. Discharged after 13 days in SCBU.

Assessment of the recording
4 hrs recording with normal parameters until 01:03, thereafter a marked decrease in signal quality. Most difficult to assess FHR but there seems to be intermittent variable decelerations with a baseline FHR decreasing from 160 to 140 bpm. ST showed a significant T/QRS baseline rise from 0.05 to 0.15-0.19 at 01:50 (visual assessment). This rise coincided with the mother returning from a visit to the toilet!

End of first stage recording



Recording obtained during active pushing



Comments

Signs of intrapartum hypoxia in a growth retarded fetus, starting with the onset of active pushing. Both CTG and ST interpretation difficult due to poor signal quality. However, from visual analysis of ST waveforms and T/QRS ratios plotted there appears to be a significant rise in baseline T/QRS exceeding 0.10 units as the mother returns from her visit to the toilet and with onset of active pushing 1h 24 min before delivery.

Case OEH 371

Date of delivery: [illegible]

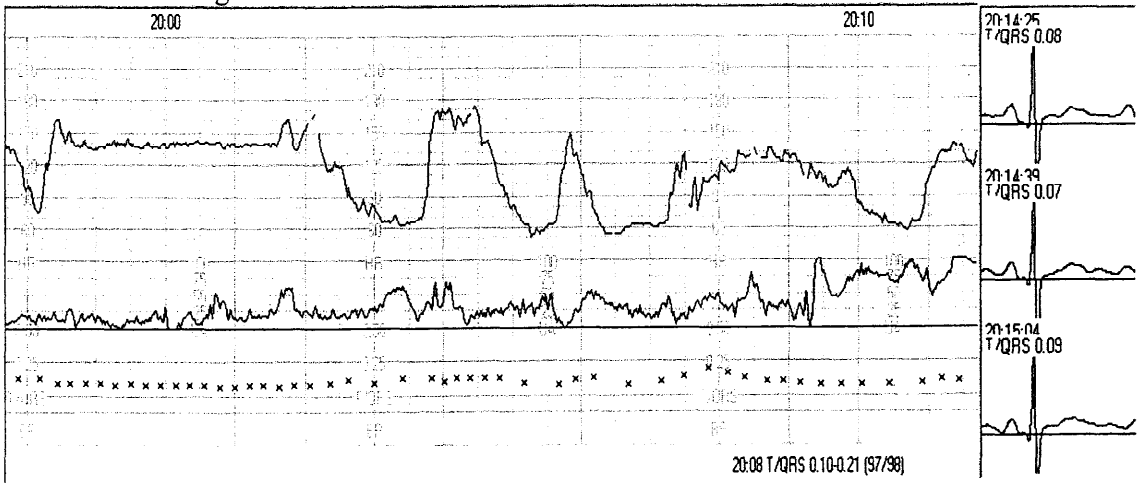
Clinical data
Para 0, normal pregnancy, spontaneous onset of labor at 39weeks of gestation
Clear liquor.
Active pushing at 23:10
NVD at 23:40

Neonatal data
Male 2710g
Apgar 3-3-10
No cord blood gas analysis obtained.

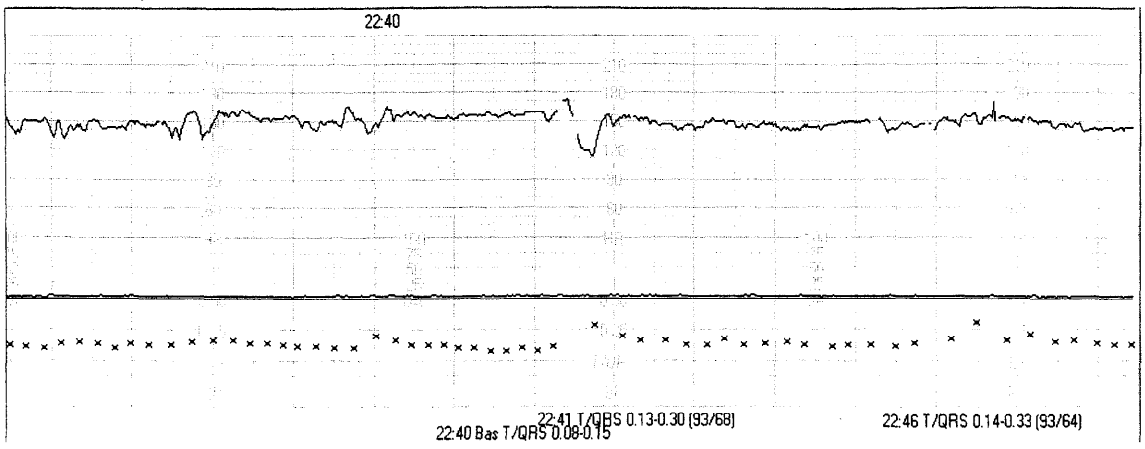
Neonatal outcome
The neonate required assisted ventilation by facemask until 6-7 min of age, rapid recovery thereafter but marked peripheral vasoconstriction. Venous lactate 11 mmol/l at 30-45 min of age. Uncomplicated stay in SCBU for 1½ days. No buffering required.

Assessment of the recording
3h 48 min of recording finishing 8 min before delivery at 23:33.
Abnormal CTG at onset of recording with deep combined decelerations soon accompanied by a significant T/QRS rise, both baseline and episodic. The situation continued with variable decelerations and episodic T/QRS rises. At 22:46 a significant baseline T/QRS rise from 0.14 to 0.33, associated with tachycardia, lowered variability but only small uncharacteristic decelerations.

Onset of recording



2.5 hrs later



Comments

A baby affected immediately at birth, with a marked lactate rise, but recovering soon thereafter. The ST waveform changes indicated a fetus at risk of hypoxia but still adequately responding to the stress of labour. Revealing the ST event log would have alerted the physician to intervene already at 20:08, i.e. 3.5 h before delivery.

Case MAD 342

Date of delivery: [illegible]

Clinical data
Para 0, uncomplicated pregnancy, induced at 42 weeks of gestation
Oligohydramnios with meconium stained liquor
FBS at 17:30, pH 7.28
Active pushing commenced at 20:15.
NVD at 20:44

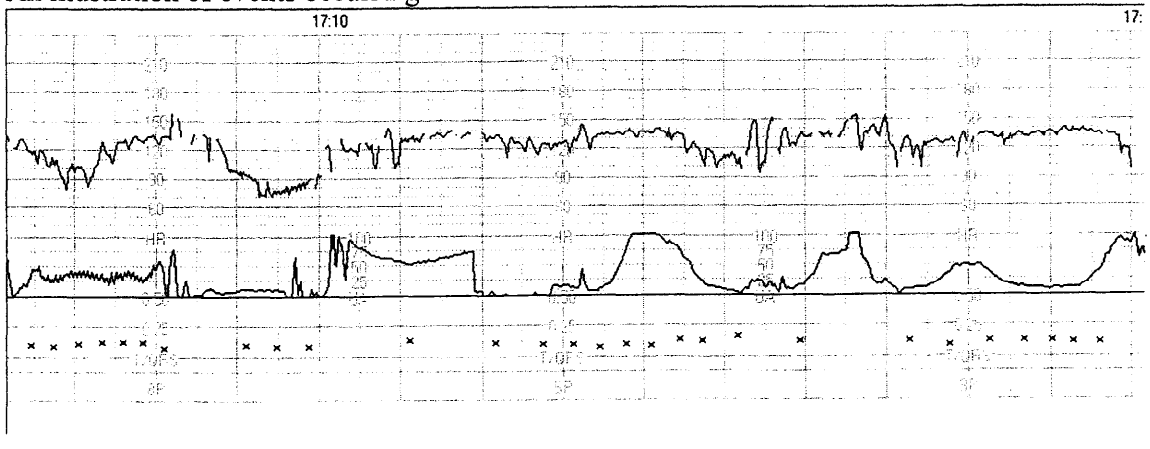
Neonatal data
Female 3325 g
Apgar 5-7-8
Cord artery: pH 6.97
PCO_2 8.08 kPa
BDecf 15.8 mmol/l

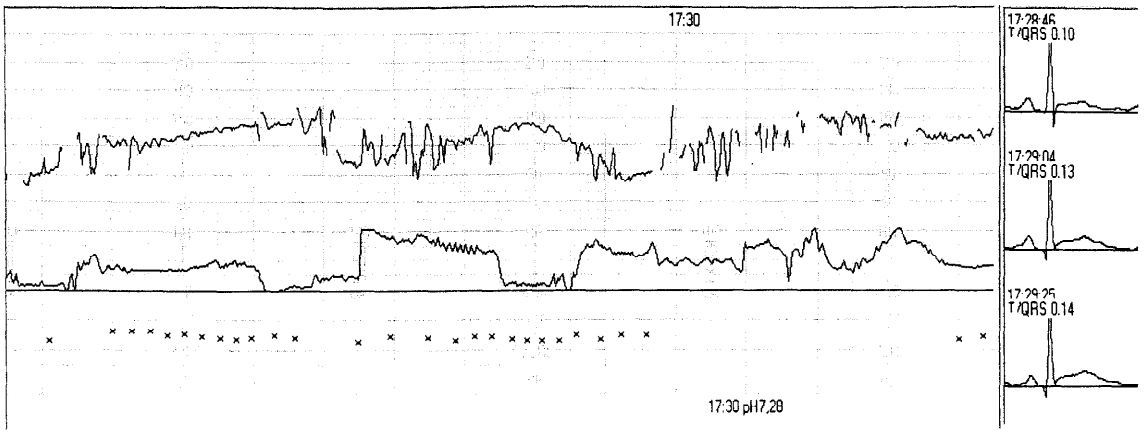
Cord vein: pH 7.04
PCO_2 5.76
BDecf 16.8

Neonatal outcome
To NICU for initial respiratory problems with metabolic acidosis, buffers given and CPAP treatment during the first couple of hours.
Otherwise, uncomplicated 1 day stay in SCBU.

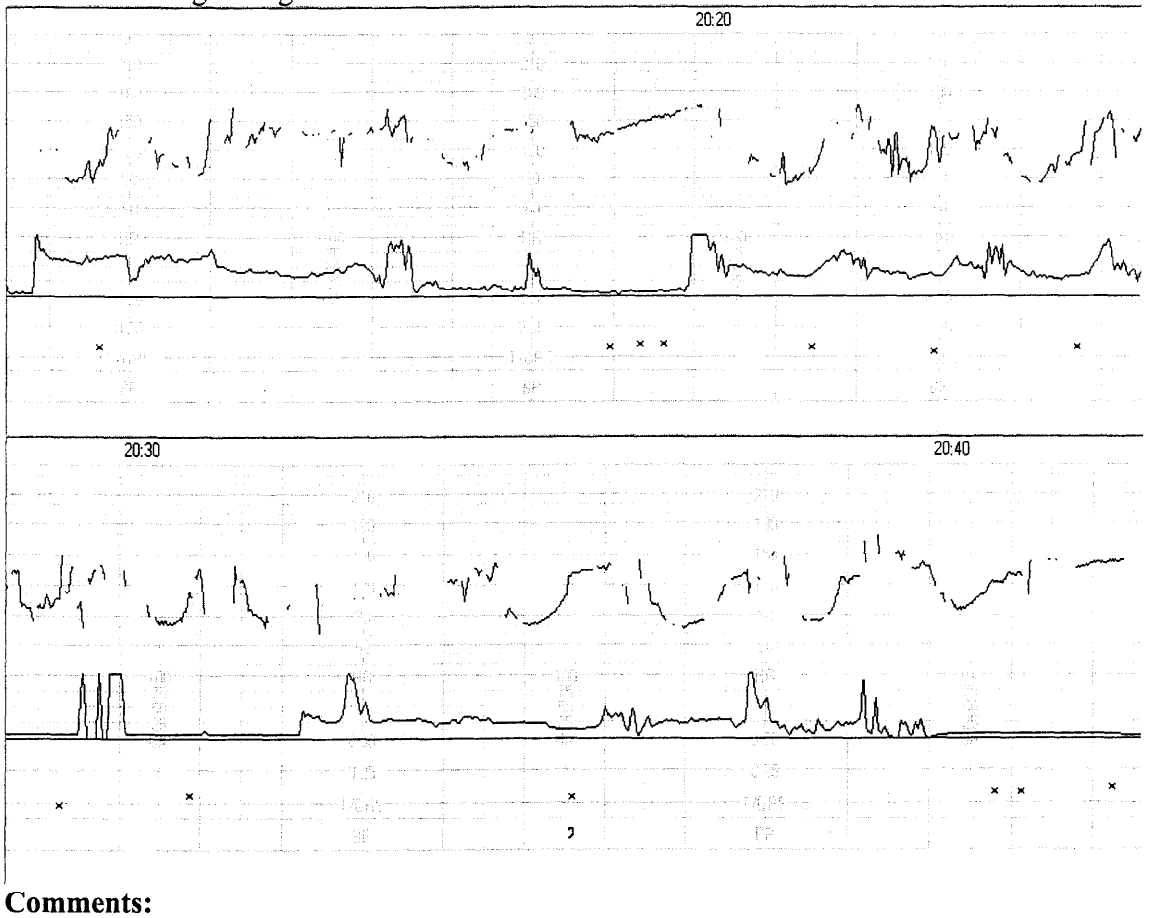
Assessment of the recording
7 hours of recording finishing 3 min before delivery. Normal reactive CTG initially. Baseline FHR approx 140 bpm. Combined decelerations occurred between 16:30 and 18:30. Last 30 min with active pushing, late decelerations.
Good quality ST data with an uneventful ST trace until last 30' when signal quality became too poor for detailed assessment. However no indications of baseline T/QRS rise.

An illustration of events occurring at FBS





STAN recording during the last 30 minutes



Comments:

A case delivered post term with oligohydramnios, cord metabolic acidosis, not requiring active resuscitation. Short lasting respiratory symptoms responding to buffering and CPAP. Unfortunately, the poor signal quality did not allow for a detailed ST assessment during the last 30 min. However, nothing to indicate a baseline T/QRS rise but signs of a lowering of the ST segment during 2nd stage. CTG changes showed late decelerations as signs of impending hypoxia. The neonatal condition, with a rapid recovery, did not indicate a substantial fetal distress.

Case MAD 438

Date of delivery: [illegible]

Clinical data
Para 2, Oligohydramnios, induced labour after 42 weeks of gestation
Active pushing commenced at 05:15
Mid cavity ventouse for failure to progress at 05:59

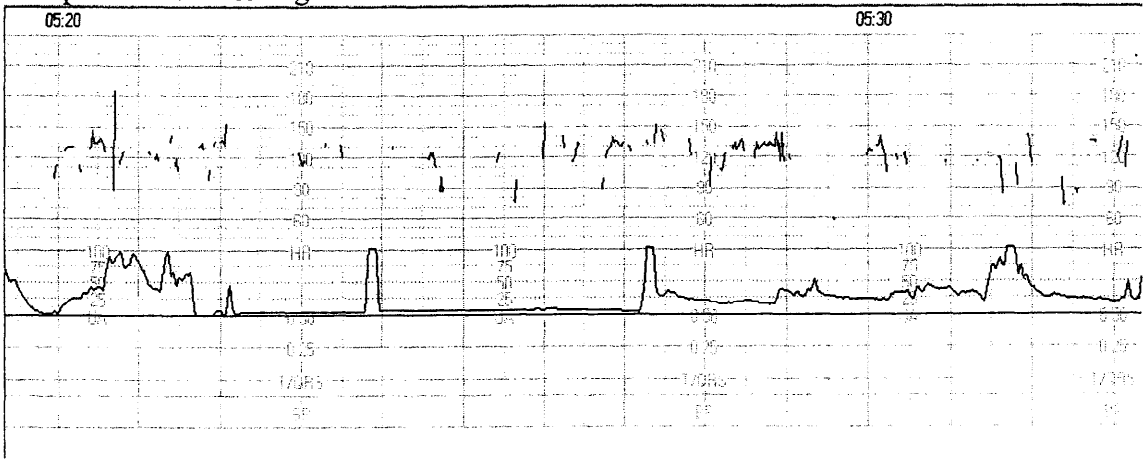
Neonatal data
Male 3660 g
Apgar 4-6-7
Cord artery: pH 6.97
PCO_2 10.4 kPa
BDecf 12.2 mmol/l

Cord vein: pH 7.17
PCO_2 6.51 kPa
BDecf 9.7 mmol/l

Neonatal outcome
Admitted to SCBU for surveillance with extra oxygen and buffering, short lasting RDS. Uncomplicated neonatal period. 1 day visit to SCBU.

Assessment of the recording
Normal CTG+ST data from onset of recording at 03:35 until 05:00 thereafter the signal quality deteriorated and poor CTG and ST information was provided.

Last part of the recording



Comments
No accurate information available during the last hour of labour in a case where ST information was blinded and the staff apparently accepted the heart rate trace despite a very poor quality.